UNITED STATES DISTRICT COURT
FOR THE WESTERN DISTRICT OF PENNSYLVANIA

GIANT EAGLE, INC.,	)	
	)	
Plaintiff,	)	Civil Action No.
	)	
v.	)	
	)	
PFIZER INC., PFIZER IRELAND PHARMACEUTICALS, WARNER-LAMBERT COMPANY, WARNER-LAMBERT COMPANY LLC, RANBAXY INC., RANBAXY PHARMACEUTICALS, INC., AND RANBAXY LABORATORIES LIMITED,	)	
	)	
Defendants.	)	

COMPLAINT

Plaintiff Giant Eagle, Inc. ("Giant Eagle") brings this civil action against Defendants Pfizer, Inc., Pfizer Ireland Pharmaceuticals, Warner-Lambert Company, Warner Lambert Company LLC, Ranbaxy, Inc., Ranbaxy Pharmaceuticals, Inc. and Ranbaxy Pharmaceuticals Limited under the antitrust laws of the United States. For its Complaint, Giant Eagle alleges as follows:

I. INTRODUCTION

1. This is an antitrust action under Sections 1 and 2 of the Sherman Act, 15 U.S.C. §§ 1-2, and Ohio's Valentine Act, Ohio Rev. Code §§ 1331.01, *et seq*., seeking overcharge damages for the delayed entry of generic versions of the historically popular brand-name drug Lipitor (atorvastatin calcium). Although the original compound patent for Lipitor expired March 24, 2010, generics remained foreclosed until November 30, 2011, over 20 months later. Pfizer[1]

[1] "Pfizer" or "the Pfizer Defendants" are Pfizer Inc., Pfizer Ireland Pharmaceuticals, Warner-Lambert Company, and Warner-Lambert Company LLC.

illegally caused this delay by implementing an overarching anticompetitive scheme, which included:

(a) perpetrating fraud upon the United States Patent and Trademark Office ("PTO") to procure Patent No. 5,273,995 (the '995 patent), which Pfizer thereafter wrongfully listed in the United States Food and Drug Administration's ("FDA") "Orange Book";

(b) filing a sham "Citizen Petition" with FDA;

(c) entering into an anticompetitive and unlawful "reverse payment" settlement agreement with defendant Ranbaxy under cover of a non-justiciable lawsuit,[2] pursuant to which Ranbaxy agreed not to launch (or permit other companies to launch) a generic version of Lipitor in the United States until November 30, 2011 in exchange for various consideration from Pfizer (the "Delay Agreement");

(d) thwarting other generic companies' efforts to obtain judicial declarations that Pfizer's various unasserted patents were invalid, unenforceable and/or would not be infringed by generic Lipitor, in order to avoid the triggering of Ranbaxy's anticipated 180-day first-to-file marketing exclusivity and thereby sustain Pfizer's and Ranbaxy's ability to, in concert, block other generic companies from launching generic Lipitor earlier than November 30, 2011; and

(e) once generic entry was imminent, entering into arrangements with pharmacy benefit managers ("PBMs") that significantly reduced the benefits of generic competition to Giant Eagle and other purchasers, thereby slowing Pfizer's loss of monopoly power with respect to those purchasers and requiring Giant Eagle to

[2] "Ranbaxy" refers to Ranbaxy Inc., Ranbaxy Pharmaceuticals Inc., and Ranbaxy Laboratories Limited.

buy more high-priced branded Lipitor, and less low-priced generic atorvastatin, than it otherwise would have bought.

2. The scheme worked as planned. Generic Lipitor was not sold until on or about November 30, 2011, far later than it would have been sold absent Defendants' illegal, anticompetitive conduct. Even after it became available, Pfizer's efforts to suppress generic substitution and postpone the loss of its monopoly power forced Giant Eagle to continue to buy branded Lipitor in higher quantities than would have occurred absent those efforts, resulting in additional harm to Giant Eagle and to the competitive process.

3. Because of Defendants' scheme to delay and suppress generic Lipitor competition, in whole or in part, Giant Eagle paid tens of millions of dollars more for atorvastatin calcium than it would have paid absent such conduct.

II. JURISDICTION AND VENUE

4. This action arises under sections 1 and 2 of the Sherman Act, 15 U.S.C. §§ 1 & 2, section 4 of the Clayton Act, 15 U.S.C. §15(a) and the Ohio Valentine Act, to recover treble damages, costs of suit and reasonable attorneys' fees for the injuries sustained by Giant Eagle as a result of Defendants' unlawful foreclosure of the United States market for atorvastatin calcium. The Court has subject matter jurisdiction under 28 U.S.C. §§ 1331 and 1337(a), and 15 U.S.C. §15.

5. Defendants transact business within this district, and they carry out interstate trade and commerce, in a substantial part, in this district and/or have an agent and/or can be found in this district. Venue is appropriate within this district under section 12 of the Clayton Act, 15 U.S.C. § 22, and 28 U.S.C. §1391(b) and (c) and 28 U.S.C. § 1407.

III. THE PARTIES

6. Plaintiff Giant Eagle, Inc. ("Giant Eagle") is a Pennsylvania corporation with its principal offices located at 101 Kappa Drive, Pittsburgh, PA. Giant Eagle is a parent company of the Tamarkin Company ("Tamarkin"), an Ohio corporation, and Riser Foods Company ("Riser"), a Delaware corporation. Giant Eagle, together with its wholly owned subsidiaries Tamarkin and Riser, is engaged in the retail supermarket and pharmacy business and owns, operates, and licenses more than 211 pharmacies, including 111 pharmacies in Ohio. Giant Eagle purchases substantial quantities of pharmaceutical products and other goods for resale to the public.

7. During the relevant period of time, Giant Eagle purchased Lipitor from wholesaler McKesson Corporation ("McKesson"). McKesson purchased Lipitor directly from Pfizer, Inc. and has assigned to Giant Eagle the antitrust claims arising out of McKesson's purchases of Lipitor that were subsequently resold to Giant Eagle. Giant Eagle brings this action in its own right and as the assignee of McKesson.

8. Defendant Pfizer, Inc. is a Delaware corporation having its principal place of business at 235 East 42nd Street, New York, New York 10017. At all relevant times, defendant Pfizer, Inc. sold branded Lipitor to McKesson and engaged in the conduct challenged in this case and attributed to the Pfizer Defendants itself and/or through its various employees and/or other agents acting within the course and scope of their duties and/or with actual, apparent or ostensible authority in connection therewith.

9. Defendant Pfizer Ireland Pharmaceuticals, formerly known as Warner Lambert Export, Ltd., is a partnership organized and existing under the laws of Ireland, with registered offices at Potter Road, Dun Laoghaire, Co. Dublin, Ireland. Pfizer Ireland Pharmaceuticals, a wholly-owned indirect subsidiary of defendant Pfizer, Inc., is the exclusive licensee of the '995

Patent and other patents. At all times relevant, defendant Pfizer Ireland Pharmaceuticals engaged in the conduct challenged in this case and attributed to the Pfizer Defendants, itself and/or through its various employees and/or other agents acting within the course and scope of their duties and/or with actual, apparent, or ostensible authority in connection therewith.

10. Defendant Warner-Lambert Company is a corporation formerly organized under the laws of the State of Delaware with offices for service of process at 235 East 42nd Street, New York, New York 10017. In 1997, Warner-Lambert and Pfizer began co-promotion of Lipitor. On June 19, 2000, Pfizer completed its merger with Warner-Lambert, whereby Pfizer purchased all outstanding shares of Warner-Lambert common stock. Each share of Warner-Lambert stock was converted into 2.75 shares of Pfizer common stock. The merger qualified as a tax-free reorganization and was accounted for as a pooling of interests. Warner-Lambert Company became a wholly-owned subsidiary of Pfizer, Inc. At the end of 2002, Warner-Lambert Company became a Delaware limited liability company and changed its name to Warner-Lambert Company LLC. Warner-Lambert Company knowingly controlled all activities of the applicant before the PTO in connection with the prosecution of the '995 Patent and other patents.

11. Warner-Lambert Company and Warner-Lambert Company LLC are collectively referred to as "Warner-Lambert." "Warner-Lambert" includes, but is not limited to, Warner-Lambert employees Bruce D. Roth, Joan Thierstein, and Jerry F. Janssen.

12. Together, the Defendants identified in paragraphs 8 through 11 are referred to herein as "Pfizer" or the "Pfizer Defendants."

13. Defendant Ranbaxy, Inc. is a Delaware corporation with a place of business located at 600 College Road East, Princeton, New Jersey 08540.

14. Defendant Ranbaxy Pharmaceuticals, Inc. is a Delaware corporation with a place of business at 600 College Road East, Princeton, New Jersey 08540.

15. Defendant Ranbaxy Laboratories Limited is a corporation organized and existing under the laws of India, with a principal place of business located at Plot 90, Sector 31, Gurgaon-122001 (Haryana), India.

16. At all relevant times, defendants Ranbaxy Inc. and Ranbaxy Pharmaceuticals Inc. acted in their own right and as agents of defendant Ranbaxy Laboratories Limited.

17. Together, the Defendants identified in paragraphs 13 through 15 are referred to herein as "Defendant Ranbaxy" or "Ranbaxy."

18. The term "Defendants" refers to all the defendants.

19. All of the Defendants' actions described in this Complaint are part of, and were in furtherance of, the illegal monopolization and restraint of trade alleged herein, and were authorized, ordered, or done by the Defendants' various officers, agents, employees, or other representatives while actively engaged in the management of the Defendants' affairs, within the course and scope of their duties and employment, and/or with the actual, apparent, and/or ostensible authority of Defendants.

IV. LEGAL BACKGROUND

A. The regulatory structure for approval of generic drugs and substitution of generics for brand name drugs

20. Under the Federal Food, Drug, and Cosmetic Act ("FDCA"), manufacturers who create a new drug product must obtain the approval of the FDA to sell the new drug by filing a New Drug Application ("NDA"). 21 U.S.C. §§ 301-392. An NDA must include submission of specific data concerning the safety and effectiveness of the drug, as well as any information on applicable patents. 21 U.S.C. § 355(a), (b).

21. When the FDA approves a brand name manufacturer's NDA, the brand manufacturer may list any patents that the brand manufacturer believes could reasonably be asserted against a generic manufacturer who makes, uses, or sells a generic version of the brand name drug prior to the expiration of the listed patents in the FDA's book of Approved Drug Products with Therapeutic Equivalence Evaluations, commonly referred to as the "Orange Book." Patents issued after NDA approval may be listed within 30 days of issuance. 21 U.S.C. §§ 355 (b)(1) & (c)(2).

22. The FDA relies completely on the brand name manufacturer's truthfulness about patents' validity and applicability; the FDA has neither the authority nor the resources to check the manufacturer's representations for accuracy or trustworthiness.

1. The Hatch-Waxman Amendments

23. The Hatch-Waxman Amendments enacted in 1984 simplified the regulatory hurdles for prospective generic manufacturers by eliminating the need for them to file lengthy and costly NDAs. *See* Drug Price Competition and Patent Term Restoration Act, Pub. L. No. 98-417, 98 Stat. 1585 (1984). A generic manufacturer seeking approval to sell a generic version of a brand name drug may now file an abbreviated new drug application (ANDA). ANDAs rely on the scientific findings of safety and effectiveness included in the brand name drug manufacturer's original NDA, but must show that the generic drug contains the same active ingredient(s), dosage form, route of administration, and strength as the brand name drug – that is, that the generic drug is bioequivalent to the brand name drug. The FDA assigns generic drugs that are bioequivalent to branded drugs an "AB" rating.[3]

[3] Generic manufacturers can also seek approval of non-AB-rated generics. The FDCA permits "hybrid" applications that are neither full NDAs containing safety and efficacy data, nor ANDA applications showing that the proposed product is the "same" as the NDA product. 21 U.S.C. § 505(b)(2). Drug products approved under this section use a safe and effective active pharmaceutical ingredient, but modify the drug product in some way so that it differs from the original NDA product, either in dosage form, strength, route of administration, formulation, dosing

24. The FDCA and Hatch-Waxman Amendments operate on the presumption that bioequivalent drug products containing identical amounts of the same active ingredients in the same route of administration and dosage form, and meeting applicable standards of strength, quality, purity and identity, are therapeutically equivalent and may be substituted for one another. Thus, for drugs that are intended to be absorbed into the bloodstream, bioequivalence demonstrates that the active ingredient of the proposed generic drug would be present in the blood of a patient to the same extent and for the same amount of time as the branded counterpart. 21 U.S.C. § 355(j)(8)(B).

25. Throughout the Hatch-Waxman Amendments, Congress sought to expedite the entry of legitimate (non-patent infringing) generic competitors, thereby reducing healthcare expenses nationwide. Congress also wanted to protect pharmaceutical companies' incentives to create new and innovative products.

26. The Hatch-Waxman Amendments achieved both goals, substantially advancing the rate of generic product launches, and ushering in an era of historic high profit margins for brand name pharmaceutical companies.

2. Paragraph IV Certifications

27. To obtain FDA approval of an ANDA, a generic manufacturer must certify that the generic drug addressed in its ANDA will not infringe any patents listed in the Orange Book. Under Hatch-Waxman, a generic manufacturer's ANDA must contain one of four certifications:

(i) that no patent for the brand name drug has been filed with the FDA (a "Paragraph I certification");

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regimen, or indication. These non-AB-rated generics are not bioequivalent to the innovator product. *See* 21 CFR 314.54.

(ii) that the patent for the brand name drug has expired (a "Paragraph II certification");

(iii) that the patent for the brand name drug will expire on a particular date and the generic company does not seek to market its generic product before that date (a "Paragraph III certification"); or

(iv) that the patent for the brand name drug is invalid or will not be infringed by the generic manufacturer's proposed product (a "Paragraph IV certification").

28. If a generic manufacturer files a Paragraph IV certification, a brand name manufacturer has the ability to delay FDA approval of an ANDA simply by suing the ANDA applicant for patent infringement. If the brand name manufacturer initiates a patent infringement action against the generic filer within 45 days of receiving notification of the Paragraph IV certification, the FDA may not grant final approval to the ANDA until the earlier of (a) the passage of 30 months, or (b) the issuance of a decision by a court that the patent is invalid or not infringed by the generic manufacturer's ANDA. The FDA may grant "tentative approval," but cannot authorize the generic manufacturer to go to market.

29. As an incentive to spur generic companies to seek approval of generic alternatives to branded drugs, the first generic manufacturer to file an ANDA containing a Paragraph IV certification gets a period of protection from competition with other generic versions of the drug. For Paragraph IV certifications made prior to December 2003, the first generic applicant is entitled to 180 days of market exclusivity. Meaning, the first approved generic is the only available generic for at least six months.

30. The ability of brand name manufacturers to delay FDA approval of an ANDA for up to 30 months merely by filing suit upon receipt of notice of a paragraph IV certification is a strong incentive to brand name manufacturers to list patents in the Orange Book – even if such patents are not eligible for listing – and sue any generic competitor that files an ANDA with Paragraph IV certifications – even if the competitor's product would not actually infringe the listed patent(s).

B. The Benefits of Generic Drugs

31. Typically, AB-rated generics cost much less than their branded counterparts. Over time, as more generic equivalents compete with each other, prices decline even further. Since passage of the Hatch-Waxman Amendments, every state has adopted substitution laws that either require or permit pharmacies, like those operated by Giant Eagle, to substitute AB-rated generic equivalents for branded prescriptions unless the prescribing physician has specifically ordered otherwise.

32. Once a generic equivalent hits the market, the generic quickly overtakes sales of the branded drug. More than 90 percent of prescriptions for drugs that are available in both branded and generic forms are filled with a generic. The speed with which generic drugs take over the market appears to be increasing: in a sample of drugs losing patent protection between 1991 and 1993, generics on average held a 44 percent market share after one year; by 2008, generic versions could capture as much as 86 to 97 percent of the market within the first month.

33. Branded manufacturers are well aware of generics' steady erosion of their previously monopolized market. Branded manufacturers thus seek to extend their monopoly for as long as possible, sometimes resorting to any (illegal) means possible. Frequently, branded manufacturers engage in tactic commonly known as "pay-for-delay" or "reverse-payment"

settlements, whereby a branded manufacturer pays its would-be generic competitor a substantial sum of money to drop its challenge to a patent and delay marketing its drug.

34. In *In re: K-Dur Antitrust Litig.*, Nos. 10-2077, 10-2078, 10-2079, 10-4571 (3d Cir. July 16, 2012) ("*K-Dur*"), the United States Court of Appeals for the Third Circuit held that such "pay-for-delay" or "reverse-payment" settlements should be considered "*prima facie* evidence of an unreasonable restraint of trade." The presumption of illegality can only be rebutted by a showing that the payment (1) was for a purpose other than delayed entry, or (2) offers some pro-competitive benefit.

V. FACTS

A. Statins

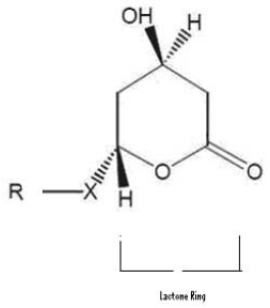
35. Lipitor belongs to a class of drugs called statins. Discovered in the 1970s, statins lower cholesterol by inhibiting the liver enzyme 3-hydroxy 3-methylglutrayl-coenzyme A reductase ("HMG-CoA reductase"). HMG-CoA reductase controls the rate of the metabolic production of cholesterol; inhibiting HMG-CoA reductase inhibits the production of cholesterol. Common thinking is that high cholesterol is associated with coronary heart disease and atherosclerosis in some populations.

36. Efforts to reduce cholesterol levels are a big business: by 1997, five of the largest pharmaceutical companies marketed and sold six different brand name statins. In 2002, almost one in ten Americans aged 20 and older took a statin. In 2004, sales of statins topped $15.5 billion, comprising 6.6% of all prescription drug sales.

37. Branded statins cost between $2.50 and $5.00 for a single daily pill ($75 to $150 a month, $900-$1,800 a year); generic statins cost markedly less, sometimes less than $1 a day.

38. Statins consist of three structural parts: a lactone ring, a linkage group (denoted as "X"), and a group or groups connected to the linkage group (referred to herein as an "R group").

Figure 1: Generalized Structure of Statins[4]



39. The R group for the well-known statins can contain one or more single rings or fused rings, and other substituent groups.

40. The lactone ring, on the right hand side, contains five carbon atoms and one oxygen atom.[5] Attached to the ring and denoted as =O is an additional oxygen called a *ketone.* The lactone ring has two major substituents: a hydroxyl group (-OH) shown at the top of the ring, and the linkage group, X, attached to the R group. The two major substituents in the lactone ring are in a *trans* position; that is, the hydroxyl group is above the plane of the lactone ring and the linkage group X is below the plane of the lactone ring.

41. In the 1970s, researchers discovered that mevastatin, naturally occurring in red yeast and rice, inhibited cholesterol synthesis.

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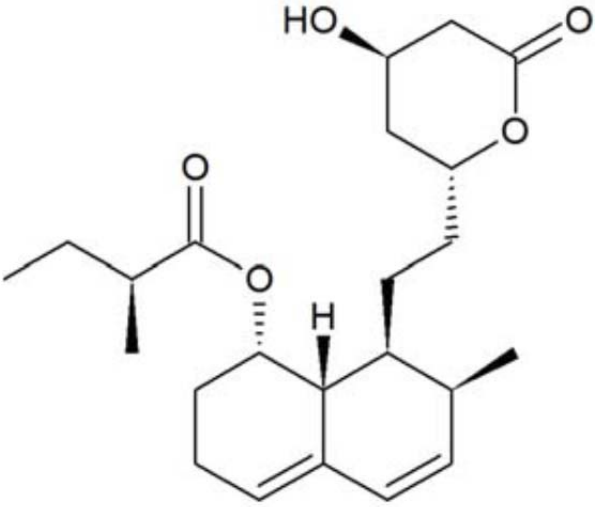
4 The three-dimensional structure of molecules can be represented pictorially in two dimensions using the following symbols to represent the orientation of the atoms in space: ◣ (solid wedge) indicates that the molecule is projecting out of the page; ⁻ (dashed wedge) indicates that the molecule is projecting behind the page; — (solid line) indicates that the molecule is in the plane of the paper.

5 The lactone ring members are shown with the chemical convention that omits the carbon and some hydrogen atoms and shows only the bonds between the carbons and other atoms. Each carbon atom is designated as the point where two bond lines connect and each carbon is assumed to have two hydrogen atoms attached.

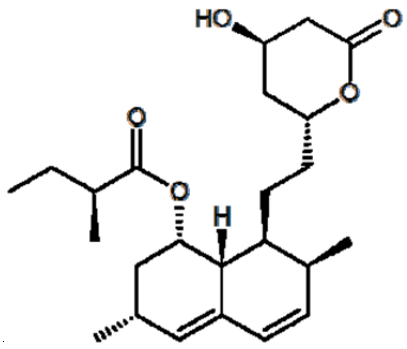
Figure 2: Mevastatin



42. Mevastatin contains the lactone ring as shown in Figure 1 (top right of Figure 2), a linkage group, X (shown as ), and an R group of two fused rings with substituents. One of the fused rings contains a methyl group (-CH_3, shown as ) on the right ring and an additional O-linked substituent group on the left ring.

43. Around the same time, researchers discovered lovastatin, naturally occurring in red yeast rice and oyster mushrooms, was another highly potent HMG-CoA reductase inhibitor. Merck sought and gained approval for Mevacor, a brand name version of lovastatin and the first statin available in the United States, in the early 1980s.

Figure 3: Lovastatin



44. The structure of lovastatin is similar to mevastatin. Lovastatin contains a lactone ring and a fused-ringed group joined to the lactone ring by a linkage group. The R group contains the same fused rings with same O-linked substituent group on the left ring and a methyl group on the right ring as found on mevastatin. Lovastatin has an additional methyl group.

45. In the early 1980s, Warner-Lambert sought to develop a "me-too" version of the already-identified statins. Researchers at Warner-Lambert came up with a formulation that used the same lactone ring as mevastatin and lovastatin but contained different linked substituents. Warner-Lambert called their new statin "atorvastatin."

B. 1986-1987: Warner-Lambert obtains the original Lipitor patent

46. On May 30, 1986, Warner-Lambert filed a patent application for a group of compounds and pharmaceutical compositions useful as hypercholesterolemic and hypolipidemic agents.[6] The patent application was entitled *"Trans-6-[2-(3- or 4-Carboxamido-Subsituted Pyrrol-1-yl)alkyl]-4-Hydroproxypyran-2-one Inhibitors Of Cholesterol Synthesis*."

47. This application would eventually lead to the issuance of the '893 patent.

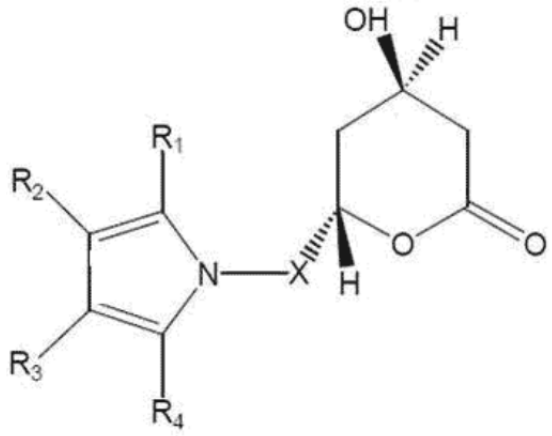
48. As this lawsuit involves how Warner-Lambert fraudulently obtained another Lipitor patent *after* the issuance of the '893 patent, and because this lawsuit involves how Warner-Lambert fraudulently avoided the prior art contained in the '893 patent, this Complaint now describes the background, claims and uses of the '893 patent, or "original Lipitor patent."

[6] The application was in the name of Bruce D. Roth. Roth was, at all relevant times, a leader of the drug discovery team at Warner-Lambert that developed Lipitor. He is the named inventor of Patent Nos. 4,681,893 and 5,273,995; the patent applicant for Patent Nos. 4,681,893 and 5,273,995; and the patent applicant in connection with the re-issuance proceedings for Patent No. 5,273,995. Patent Nos. 4,681,893 and 5,273,995 issued to Roth and were assigned to his employer, Warner-Lambert. Roth is not individually named as a defendant in this action.

1. The patent specification claims atorvastatin, "the corresponding ring-opened acids derived therefrom" in salt form, and the R-trans and S-trans enantiomers

49. Warner-Lambert stated in the patent specification for the Original Lipitor Patent that in its broadest aspect the present invention provides compounds of structural formula I.

Figure 4: Warner-Lambert's Structural Formula I



50. Like other statins, structural formula I contains a lactone ring, a linkage group (X), and an R group.

51. Warner-Lambert claimed the disclosed compounds were useful as hypocholesterolemic or hypolipidemic agents by virtue of their ability to inhibit the biosynthesis of cholesterol through inhibition of the HMG-CoA reductase enzyme. For support, the specification detailed the biological activity of three compounds compared to the prior art.

52. Research in the 1980s demonstrated that the open lactone ring forms of statin molecules were highly potent cholesterol synthesis inhibitors and are often more potent than the closed lactone ring forms of the same molecules. Warner-Lambert claimed that the invention contemplated the hydroxyl acids, or structural formula I with an opened lactone ring:

> Also contemplated as falling within the scope of the present invention are the hydroxy acids, and pharmaceutically acceptable salts thereof, derived from the opening of the lactone ring of the compounds of structural formula I above.

53. Importantly, *Warner-Lambert's patent application specifies and covers a compound in which the R-trans enantiomer is isolated*:

> The compounds of structural formula I above possess two asymmetric carbon centers, one at the 4-hydroxy position of the pyran-2-one ring, and the other at the 6-position of the pyran-2-one ring where the alkylpyrrole group is attached. This asymmetry gives rise to *four possible isomers***,** two of which are the R-cis- and S-cis-isomers and the other two of which are the R-trans- and S-trans-isomers. *This invention contemplates only the trans-form* of the compounds of formula I above.

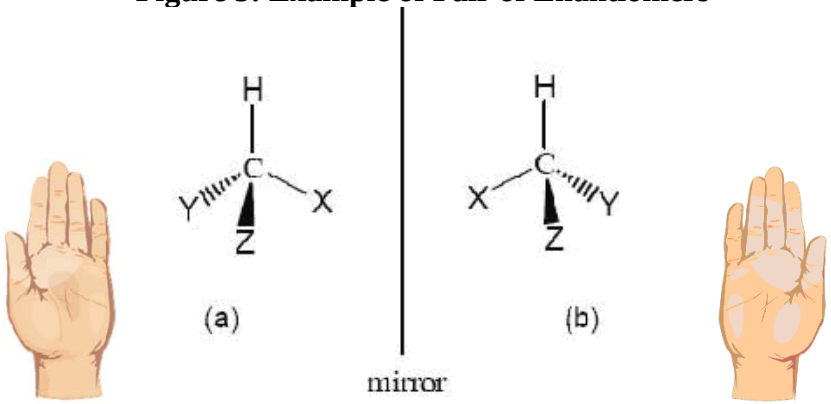
(Emphases added.)

2. The chemistry of enantiomers

54. To understand how the '893 patent covered compounds which included the isolated "R-trans enantiomer," and that it included the R-trans enantiomer in calcium form, some background of the chemistry of enantiomers is helpful. (Before doing so, it is noted that Pfizer does not dispute the patent coverage of the original Lipitor patent for atorvastatin calcium, i.e., a compound having the structural formula I, which included versions in which the R-trans enantiomer is isolated; again, this description provides the context for Defendants' later fraudulent actions.)

55. *Enantiomers* are isomers that are mirror images of each other but cannot be superimposed. For example, a person's left hand and right hand are non-superimposable mirror images of each other. Images (a) and (b) in Figure 5 below are enantiomers (where the carbon atom is the chiral center around which a compound's structure is built).

Figure 5: Example of Pair of Enantiomers



56. Pairs of enantiomers share many chemical and physical properties, such as identical melting points, solubility, and colors. Other properties, such as biological properties, may be vastly different.

57. Enzymes, including HMG-CoA reductase, typically display a preference for interacting with one enantiomer over the other. It is common for one enantiomer of an enantiomeric pair to have all or most of the biological activity when interacting with an enzyme, while the other has little or no biological activity.

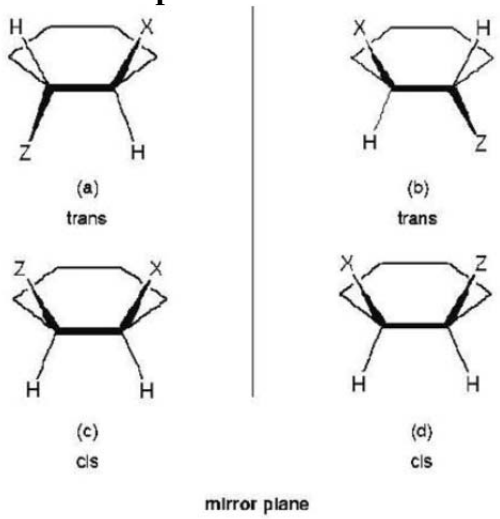
58. Enantiomers can be distinguished from one another by their effect on the rotation of polarized light, and are said to be *optically active.* Enantiomers reflect polarized light in either a clockwise direction (right, denoted "+") or a counter-clockwise direction (left, denoted "-"). An unequal mixture of two enantiomers is optically active; the degree of optical rotation reflects the percentage of each enantiomer in the mixture. When equal mixtures of two opposite enantiomers are present, called a *racemic mixture* or *racemate,* the mixture is not optically active because the optical rotations of the enantiomers cancel each other.

59. To differentiate enantiomers in written form, each enantiomer is assigned a configuration, i.e., the arrangement of atoms that characterizes a particular enantiomer and

represents the molecule's three-dimensional structure. Configuration designations are determined by priority rules that rank the atoms or substituent group of atoms that are attached to the chiral center. If the priority proceeds in a clockwise direction, the enantiomer has an 'R' (right) configuration; if the arrangement is counter-clockwise, the enantiomer has an 'S' (left) configuration.

60. In addition to R/S and +/- configurations, a molecule's configuration can also reference the location of the substituent atoms or groups of atoms relative to each other. An arrangement where both the major substituents lie on the same side of the plane of reference is called a *cis* arrangement. An arrangement where the major substituents appear on the opposite sides of the plane is called a *trans* arrangement. The placement of X and Z in the figure below demonstrates these cis and trans arrangements.

Figure 6: Examples of Cis and Trans Arrangements



61. The lactone rings found in statins have two chiral centers, one at the carbon with the hydroxyl group and the other at the carbon attached to the linker. Rings containing two chiral centers give rise to four possible isomers – the R-cis-isomer, the S-cis-isomer, the R-trans-

isomer, and the S-trans-isomer – and two enantiomeric pairs – R-cis-isomer & S-cis-isomer and R-trans-isomer & S-trans-isomer.

62. Research at the time demonstrated that the preferred configuration for the lactone ring in a statin – the configuration offering the highest level of cholesterol inhibition – was the R-trans configuration.[7] Both mevastatin and lovastatin have lactones in the R-trans configuration. In the case of HMG-CoA reductase inhibitors, the R-trans enantiomer appeared to be the active enantiomer and the S-trans enantiomer the inactive one.

63. Consistent with this conventional thinking, Warner-Lambert's application for the '893 patent contemplated the trans-form of compounds in structural formula I, i.e., racemic or enantiomeric forms of structural formula I. Furthermore, the application contemplated atorvastatin in a variety of formulations, including calcium salts.

3. 1987: The PTO issues the original Lipitor patent

64. On July 21, 1987, the PTO issued the '893 original Lipitor patent.[8] The '893 patent was assigned to Warner-Lambert. In the absence of an extension, the original Lipitor patent would have expired on May 30, 2006, twenty years from the date of the first application. Later extensions lengthened the period of patent protection until March 24, 2010. (The extensions are discussed later.)

65. The '893 patent envisaged the ability to have just the R-trans or S-trans enantiomers of compounds of structural formula I. The '893 patent also recognized that these compounds could be in acid or salt form. While the '893 patent covered multiple formulations

[7] See, e.g., Alberts, A. *et al., J. Proc. Natl. Acad. Sci. USA* 1980, 77:3957; Stokker, G.E., *et al., J. Med. Chem.* 1985, 28:347-358; Stokker, G.E. et al., *J. Med. Chem,* 1986, 29: 849-852.

[8] The PTO conducted two separate reexamination proceedings with respect to the '893 patent. Neither of these reissue proceedings is relevant to Giant Eagle's claims in this matter.

of atorvastatin, Warner-Lambert focused on developing and commercializing the R-trans enantiomer of structural formula I in calcium salt form, which it called "atorvastatin". The '893 patent thus covered atorvastatin.

C. 1989-1993: Warner-Lambert obtains a follow-on enantiomer patent by fraud[9]

66. In April of 1989, Warner-Lambert internally designated atorvastatin as a "lead compound" for further investigation. At this time, Warner-Lambert knew that the '893 patent covered atorvastatin, and that it would provide Warner-Lambert with many years of patent protection and many years of exclusive sales of atorvastatin (later called Lipitor). Nevertheless, Warner-Lambert sought to extend *even further* the period for exclusive sales for its new statin.

67. In doing so, Warner-Lambert faced certain realities. Warner-Lambert knew that the R-trans enantiomer was the active enantiomer responsible for atorvastatin's ability to inhibit cholesterol. Warner-Lambert also knew the PTO would reject an application to patent an enantiomer covered by the '893 patent; after all, such an "invention" would be either anticipated by (that is, already covered by) the '893 patent, or be obvious in light of the '893 patent. Thus, Warner-Lambert knew it could only obtain a follow-on patent specifically for the R-trans enantiomer if it could convince the PTO that the isolated R-trans enantiomer had some surprising or unexpected characteristic.

68. Senior management at Warner-Lambert instructed the Warner-Lambert researchers to review the *pre-existing* biological data for the R-trans enantiomer to find data that

[9] Many of the facts recounted in this section have come to light during international patent litigation. *See, e.g.*, *Ranbaxy Australia Pty Ltd. v. Warner-Lambert Company LLC* (Appeal) 2008 FCAFC 82 (May 28, 2008); *Pfizer Canada Inc. v. Ranbaxy Labs. Ltd*., 2007 FC 91 (January 25, 2007); *Ranbaxy Australia Pty Ltd. v. Warner-Lambert Company LLC*, 2006 FCA 1787 (December 20, 2006); *Pfizer Canada Inc. v. Novopharm Ltd.*, 2006 FC 1471 (Dec. 7, 2006).

supported both (i) a claim that the activity of the isolated R-trans enantiomer was surprising and (ii) the patentability of the isolated R-trans enantiomer.

69. During a meeting, Warner-Lambert senior management asked what the patent coverage was for the pure R-trans enantiomer. Roth responded that the R-trans enantiomer was covered under the '893 patent. Senior management then asked whether there was anything about the pure R-trans enantiomer that would make it patentable in and of itself. But Roth didn't know of any surprising characteristics that had unfolded over his years of working with the enantiomer atorvastatin. So Don Maxwell, the vice president of discovery research, assigned Roth the task of going back to existing lab books to see if there was some data that showed something surprising. Roth was instructed to provide any surprising data to Wyeth patent attorney Joan Thierstein.

70. Regarding the instructions from these senior Warner-Lambert officials, Roth has stated,

> if I found something surprising I would provide that. And what I did do was I provided that information to the patent attorney for Warner-Lambert and asked if that was sufficient, and it was and so that was the data that was used.[10]

71. Of course, when senior Warner-Lambert management sent Roth back to the old laboratory notebooks to "find" something surprising, there was already a wealth of knowledge about statins and the formulation of isolated R-trans enantiomers. The state of the art regarding statin formulations sets a context for the later fraud.

1. Knowledge of one skilled in the art of statins in 1989

72. Statins are in the field of synthetic organic chemistry as it applies to discovery of compounds suitable for use as drugs directed to the regulation of the cholesterol biosynthetic

[10] *Ranbaxy Australia*, 2006 FCA 1787 at 267.

pathway and HMG-CoA reductase inhibitors. One of ordinary skill in the art of statins would have at least a bachelor's degree in organic or medicinal chemistry; a general working knowledge of statins; several years of bench work in organic molecule synthesis; some general knowledge of biochemistry and enzymology; knowledge of stereochemistry of pharmaceutically active compounds; and knowledge of resolving racemates.

73. In 1989, one skilled in the art would be knowledgeable about the biological pathway for the synthesis of cholesterol, including that HMG-CoA reductase is the rate limiting enzyme in the biological pathway for cholesterol produced in an organism. One skilled in the art would also know that statins were potent inhibitors of HMG-CoA reductase and that the scientific literature had described *in vitro* assays as methods for testing a compound's ability to inhibit cholesterol synthesis.

74. One skilled in the art would be aware that mevastatin (compactin) is a natural HMG-CoA reductase inhibitor that exists as a single enantiomer. One would also be aware that lovastatin (mevinolin), another potent inhibitor of HMG-CoA reductase, had been isolated and was structurally very similar to compactin. One would know that both mevastatin and lovastatin have lactones in the *R-trans* configuration.

75. One skilled in the art would also be aware that pravastatin (1979), simvastatin (1981), and fluvastatin (mid-1980s) were developed/isolated prior to 1989.

76. One skilled in the art would understand that pharmaceutical research into improved inhibitors of HMG-CoA was focused on analogues of known statins. One would be aware that researchers were retaining the lactone ring while investigating substitutions on the remainder of the molecule.

77. One skilled in the art would know that the ring-opened form of the upper lactone portion of the previously discovered statins is significantly more active in inhibiting HMG-CoA reductase than the lactone (closed-ring) form.

78. One skilled in the art would be knowledgeable that HMG-CoA reductase inhibitors are enantiomeric, and one enantiomer is likely to be more active than the other. One would know that the biological activity of a racemate in a biological system can be quite different from that of a single enantiomer, and one enantiomer is approximately twice as active as the racemate in terms of its operation in a target biological system *(*i.e., one enantiomer is the "active" isomer, while the other is "inactive," and thus the active enantiomer is about twice as active as the racemic mixture). One would also know that it is desirable to separate and remove the less active enantiomer.

79. One skilled in the art would know that in the case of HMG-CoA reductase inhibitors, the R enantiomer was very likely to be the active enantiomer and, conversely, the S enantiomer was very likely to be the inactive enantiomer. One would know that these expected activities could be known with certainty by isolating and testing the activity of the enantiomers.

80. One skilled in the art would understand that racemic mixtures can be separated or resolved into the individual enantiomers by well-known methods of separation or resolution. Similarly, one would be aware that single enantiomers can be isolated by chiral or achiral synthesis.

81. One skilled in the art would be knowledgeable that it was common practice among medicinal chemists and others working in the drug discovery field in 1989 to use a single structural formula to represent both enantiomers individually as well as mixtures of enantiomers. One would be similarly aware that whether a diagram depicting the structural form for a

molecule or class of molecules shows a particular stereochemistry configuration (whether absolute or relative) depends on the context in which the diagram appears. One would know that if a diagram of a single enantiomer was intended to depict a racemate, to the exclusion of the enantiomer, it was possible to add an additional descriptor, such as (+/-), RS, or ('rac'), which would make it clear that the structure represented only a racemate.

82. One skilled in the art, given the '893 original Lipitor patent, would have known that compounds in the structural formula I were racemic, that there were a discreet number of pure optically active enantiomers possible from the structural formula, and that there were known methods for dissolving the racemic mixture into the pure optically active isomers.

2. The application: Warner-Lambert fraudulently claims the R-Trans enantiomer is ten times more active than the racemate

83. On July 21, 1989, Warner-Lambert submitted a patent application for the optically pure active R-trans isomer, *i.e.,* for the R-trans form of the ring-opened acid described in the '893 patent: [R-(R*R*)]-2-(4-fluorophenyl)-β,δ-dihydroxy-5-(1-methylethyl)-3-phenyl-4-[phenylamino)carbonyl]-1H-pyrrole-1-heptanoic acid and "its lactone form and salts thereof." Roth was the applicant. The patent application was signed and submitted by a Warner-Lambert employee. The application would eventually lead (albeit by fraud) to the issuance of the '995 enantiomer patent.

84. Roth was the inventor and applicant; as part of the application, Roth provided a declaration acknowledging his duty to disclose information material to the examination of the application to the PTO, pursuant to 37 CFR §§ 1.56 – 1.63. Roth appointed Warner-Lambert's patent attorneys as his attorneys/agents and authorized them to prosecute the application. He further directed that all correspondence related to the patent application be sent to Warner-

Lambert attorney Joan Thierstein. The application itself was signed and submitted by a Warner-Lambert employee, Elizabeth M Anderson.

85. The application for the enantiomer patent was prosecuted from 1989 to 1993. The back and forth between Warner-Lambert and the PTO over those years demonstrates the materiality of Warner-Lambert's misrepresentations.

86. In the application, Warner-Lambert stated as fact that, "[i]t is now *unexpectedly found* that the enantiomer having the R form of [a] ring-opened acid [described in the '893 patent] . . . *provides surprising inhibition* of the biosynthesis of cholesterol."[11] Warner-Lambert further stated as fact that "an ordinarily skilled artisan may not predict the *unexpected and surprising inhibition* of cholesterol biosynthesis of the present invention in view of [prior] disclosures." In support of these claimed facts, Warner-Lambert presented only one piece of evidence: a short table (the "CSI table") stating that Warner-Lambert's Cholesterol Synthesis Inhibition ("CSI") assay data ostensibly showing the R-trans enantiomer is *one hundred-times more active* than the S-trans enantiomer, and *ten-times more active* than the racemate, in inhibiting the synthesis of cholesterol:

is now also incorporated by reference therefor. The CSI data of the compound I, its enantiomer the compound II and the racemate of these two compounds are as follows:

Compound	IC_{50} (micromoles/liter)
[R-(R*R*)] isomer	0.0044
[S-(R*R*)] isomer	0.44
Racemate	0.045

Accordingly, the present invention is the pharmaceutical composition prepared from the compound of the formula I or II or pharmaceutically acceptable salts thereof.

[11] All emphases appearing in quotations have been added, unless otherwise noted.

87. Warner-Lambert stated as fact that the "present invention" — the R-trans enantiomer — (regardless of the salt form it might take) — was ten times more powerful than its racemate in inhibiting cholesterol synthesis based on the data presented in the CSI table.

88. A "CSI assay" measures the ability of a compound to inhibit cholesterol biosynthesis along the entire cholesterol biosynthesis pathway and is one of the most commonly used methods to test a compound's ability to inhibit the synthesis of cholesterol *in vitro.* The CSI test does not identify the specific step in the cholesterol biosynthetic pathway that is being inhibited, nor is it specific to HMG-CoA reductase. The results of a CSI assay are reported as an IC_{50} value, the concentration of a test compound that produces 50% inhibition in the conversion of cholesterol-[^{14}C] acetate to radioactive cholesterol.[12]

89. One skilled in the art of statins in 1989 — and, indeed, one skilled in the art of statins today — would have expected the active R-trans enantiomer to be about twice as active as the racemate in inhibiting cholesterol synthesis. Activity of one enantiomer that is more than ten times that of the racemate would have been "unexpected" and "surprising" if the findings were based on true or accurate data. In fact, they were not.

a. The CSI table is both misleading and affirmatively false

90. Warner-Lambert's biological data — the CSI table — was both affirmatively false and intentionally presented in a misleading manner. The CSI table purports to present reliable scientific data. It does not. In truth, it contains limited data cherry-picked from multiple flawed tests conducted over several years using different formulations of various atorvastatin salts. And the biological data is false: the reliable data actually shows that the R-trans

[12] Two other commonly used methods of measuring a compound's inhibition of cholesterol are the *in vivo* Acute Inhibition of Cholesterol Synthesis ("AICS") assay and the *in vitro* CoA Reductase Inhibition ("COR") assay. The COR assay measures a compound's ability to inhibit HMG-CoA reductase specifically, but also is used to quantitatively compare the biologic activity of compounds.

enantiomer is, as expected, only about two times more active than the racemic mixture — not the tenfold increase Warner-Lambert claimed.

91. The CSI table is false and misleading because it does not present reliable data. CSI assays can vary greatly from one test to another. Warner-Lambert's CSI table does not disclose the source of its data, and fails to indicate the number of CSI assays performed, the degree of variation in the test results, what molecules were tested, the time period over which the assays were run, or whether the results presented were drawn from multiple tests. Given this lack of specificity, a skilled addressee would conclude that (1) Warner-Lambert would not have included the CSI table in the specification in such an unqualified way unless the data had been confirmed by a number of repeat assays and (2) it fairly depicted all such appropriate data.

92. Even though it is not apparent from the face of the specification, Warner-Lambert claimed in subsequent litigation that the CSI table was created by averaging the results of all of the available CSI screens. This was not true. Warner-Lambert ran a number of CSI assays prior to applying for the '893 and '995 patents — over a multi-year period and on various salt formations — as it tested the R-trans enantiomer of structural formula I. The results fluctuated wildly. Warner-Lambert cherry-picked from among the results — not using all the results — in order to generate a table that purportedly supported the claim of "surprising activity."

93. For example, the CSI table combines results from a number of different CSI assays and compares them to a separate CSI assay. But the standard in the 1980s for giving numbers of the kinds found in the CSI table was to conduct repeated head-to-head tests; Roth himself has acknowledged that head-to-head testing provides the best way to compare quantitative differences in activity. However, the data presented to the PTO for the R-trans enantiomer and S-trans enantiomer were taken from a single run of the same experiment: CSI

120. And in bizarre contrast, the data collected for the racemate represents an "average" of five separate assays: CSI 92, CSI 93, CSI 95, CSI 102, and one of three recorded values from CSI 118.

Figure 7: Source of Data Presented in CSI Table.

Compound	IC 50 (micromoles/liter)
R-trans Enantiomer	.0044
S-trans Enantiomer	.44
Racemate	.045

Source	Original Form	IC 50 (micromoles/liter)
CSI 120	Sodium Salt	.00444
CSI 120	Sodium Salt	.44
CSI 92	Lactone	.0346
CSI 93	Lactone	.0275
CSI 95	Lactone	.0631
CSI 102	Lactone	.0912
CSI 118	Sodium Salt	.0097

94. Second, taking an average across different days and experiments is not appropriate. The five "averaged" assays were conducted over a three-year period from July 1985 through October 1988. When taken as a whole, the results of these five experiments reported for the racemate are so variable that they cannot be averaged together with any reliability and do not provide a scientifically meaningful result.

95. Third, it is also inconsistent with accepted pharmaco-chemistry to "average" the results of CSI values derived from both opened lactones and separately synthesized sodium salts. Four of the assays reflected in the racemate data in the CSI table started with the lactone (unopened) form of racemic atorvastatin and were treated with sodium hydroxide in an effort to

open the lactone ring. In contrast, one of the assays started with an opened formulation of the racemic atorvastatin, the sodium salt.

96. One skilled in the art would be aware that if lactone rings do not fully open when exposed to sodium hydroxide, the presence of inactive material will result in a higher IC_{50} value – indicating the compound is less active than it actually is. One skilled in the art would also expect that the IC_{50} values for the racemic lactones in each of the four CSI assays would be similar, and not report a four-fold difference – from .02 (CSI 93) to .09 (CSI 102). One skilled in the art would also expect the IC_{50} values for the racemic lactones (if properly and fully opened) to be similar to the value of the racemic sodium salt, and not report a tenfold difference – from .009 (CSI 118) to .09 (CSI 120). Such disparate values show that not all of the lactone rings opened during the test and/or other solubility issues that compromise the accuracy of the data. The large differences were caused by solubility differences, not by the "inherent" differences in ability to retard synthesis.

97. Notwithstanding the inconsistency with accepted science of using an average value, the table does not even constitute a true average. As shown in Figure 8 below and although available, Warner-Lambert did not include all results from all conducted CSI assays, omitting the results of CSI 107, CSI 111, CSI 112, CSI 119, CSI 120, CSI 122, CSI 123, CSI 124, CSI 136, and CSI 138.

Figure 8: CSI Data

CSI#	Date	Racemic Lactone	R-trans Lactone	S-trans Lactone	Racemic Sodium Salt	R-trans Sodium Salt	S-trans Sodium Salt	Racemic Calcium Salt	R-trans Calcium Salt	S-Trans Calcium Salt
92	7/24/85	.0346								
93	8/27/85	.0275								
95	10/15/85	.0631								
102	1/15/87	.0912								
107	7/20/87		.0355	.631						
111	2/25/88							.0024		
112	3/28/88							.0776		
118*	10/24/88				.00977			.257	.0251	> 1.0
					.00913			.234	.0216	
119	11/15/88							.00324		
120	2/2/89					.00498	.444			
122	4/21/89					.00313				
123	5/31/89								.00948	
124	6/12/89				.001					
136	7/31/91					.0322				
138	1/31/95					.0169				

* = test calculated multiple values using different methods.

Blue = Roth used in CSI table

Yellow = Roth reported in the Roth Declaration (discussed *infra*)

98. Depending on which assays were included or excluded, the CSI table could have, and would have, reported very different results. For example, Roth acknowledged that had the results of CSI 107 been included in his "average," there would be no surprising or unexpected result. Rather, had CSI 107 been included, the CSI table would only show, as expected, a twofold increase in the activity of the R-trans enantiomer compared to the racemate. Roth has claimed he did not include CSI 107 because he believed that the compounds it tested were not

enantiomerically pure; yet, he included the results of CSI 120, which suffered from a not substantially different level of contamination.

99. Similarly, the CSI table would have shown only this expected twofold increase had Warner-Lambert excluded the results of CSI 118 from the "average."

100. No matter how Warner-Lambert, Roth, or others at the company rigged the numbers, the fact is that the R-trans enantiomer is only twice as active as the racemate.

b. Warner-Lambert's representations that the R-Trans enantiomer was ten times more active than the racemate were affirmatively false and misleading

101. Warner-Lambert's claim that the R-trans enantiomer has surprising activity compared to the racemate is false. The only consistent test results show that the R-trans enantiomer is, as expected, only about twice as active as the racemic mixture.

102. Warner-Lambert, including Thierstein and Roth, did not tell the PTO that it possessed data that expressly contradicted representations in its patent specifications.

103. In addition to CSI assays, Warner-Lambert assessed the activity of the R-trans enantiomer, S-trans enantiomer, and the racemate through the *in vivo* AICS assay. The AICS assay — the only screen to be conducted twice and with consistent results — showed a twofold increase in activity of the R-trans enantiomer over the racemate. Warner-Lambert's own research report, dated May 21, 1989, states that the R-trans enantiomer was approximately *twofold* more active at inhibiting cholesterol synthesis acutely *in vivo* compared to the racemic mixture, and *that this is to be expected* if 50% of the racemic salt is the inactive isomer.

104. Warner-Lambert did not submit the AICS data to the PTO.

105. A June 1, 1989 report signed by Roth also reported a twofold increase in activity of the active enantiomer over the racemate, indicating that, as expected, the R-trans calcium salt was twofold more potent than the racemic calcium salt, which contains 50% inactive isomer.

Other internal memoranda from September and December 1989 similarly conclude that, as expected, the R-trans enantiomer was twice as active as the racemate. But Warner-Lambert never shared *its own conclusion* with the PTO.

106. Warner-Lambert also assessed the activity of the R-trans enantiomer, S-trans enantiomer, and the racemate through the *in vivo* COR assay. The COR data was consistent with a twofold increase in activity of the R-trans enantiomer over the racemate. But Warner-Lambert never submitted the COR data to the PTO.

107. Roth and Warner-Lambert knew that the PTO would read the CSI table as fairly reflecting all of the appropriate CSI data available to Warner-Lambert for the relevant compounds, and assume the data as a whole provided reasonable grounds for the findings set forth in the CSI table. Roth and Warner-Lambert intended that the CSI table be read as suggesting a tenfold increase in activity and therefore supporting patentability.

108. Roth and Warner-Lambert also knew that the CSI data did *not* provide any "surprising" results. After all, Warner-Lambert scientists, including Roth, had conducted the various CSI assays over a period of more than three years. Certainly, if the assays had disclosed anything surprising — and certainly something as surprising as the finding that the isolated R-trans enantiomer would have ten-fold biological activity over the racemic mixture — that would have been learned, in real time, as the tests unfolded. But they did not. None of Warner-Lambert's internal documents (produced do date in related litigation) or any of the literature published by Roth and his team concerning the discovery of atorvastatin refers to, or even suggests, a ten-fold increase in activity.

109. Instead, it was only after senior Warner-Lambert managers (not the scientists) instructed Roth to go back and "find" something in the data, and after a hodge-podge analysis of

different tests on different compounds was cobbled together, that the claimed ten-fold biological activity materialized.

110. Furthermore, accepted chemistry in 1989 provided for conducting controlled testing of the proposed hypothesis, i.e., that there were some "surprising" attributes of the isolated R-trans enantiomer over the racemic mixture. This would have entailed, then, Warner-Lambert conducting *new* tests in response to senior management's demand to find something surprising. Instead, the entire direction, dictated by senior Warner-Lambert management, was *not* to conduct acceptable science in order to make fair and accurate representations to the PTO. The instructions were simply to go back and gin up old data to give an impression, albeit false, of some type of "surprising" attribute.

3. The initial rejection: the PTO determines the claimed compounds are anticipated by the '893 patent

111. On March 22, 1990, pursuant to 35 U. S. C. § 102(b), the PTO rejected all claims in the initial enantiomer application as anticipated by, i.e., covered by, the '893 patent. The PTO determined that the '893 Patent restricted the invention to the trans-isomers and specified the R*, R* configuration. Thus, the PTO determined that the claimed compounds, salts, compositions, and method were considered to be anticipated by the '893 patent. Put simply, the PTO rejected Warner-Lambert's enantiomer patent application because the invention was already covered by the claims in the original Lipitor patent.

112. The concepts of "anticipation" and "non-obviousness" are distinct but related concepts under patent law. A proposed invention may be rejected under 35 U. S. C. § 102(b) as being anticipated – that is, already covered by – a previous patent. Alternatively, even if a proposed invention is not identically disclosed or described as set forth in § 102, under 35 U. S. C. § 103 a patent may not issue for obviousness "if the differences between the subject matters

sought to be patented and the prior art are such that the subject matter as a whole would have been obvious at the time the invention was made to a person having ordinary skill in the art to which said subject matter pertains[.]" Because the patent examiner concluded that the '893 patent anticipated (that is, already covered) the isolated R-trans enantiomer form of atorvastatin, the examiner did not need to reach the concept of obviousness.

113. In response to this rejection, Warner-Lambert argued against anticipation on the technical grounds that the claimed compounds were for individual enantiomers and therefore differ from the teaching in the '893 patent only to mixtures of enantiomers. Warner-Lambert argued that the '893 patent did not specifically identify, and therefore did not technically "anticipate," the R-trans enantiomer. Specifically, Warner-Lambert argued that in molecules of the kind disclosed in the '893 patent, each possible isomer also exists in two forms which depend on a configuration which is expressed in absolute terms relative to the remainder of the molecule. Warner-Lambert stated that the forms are denoted as an R form and an S form, and these two forms are recognized by an ordinarily skilled artisan to be enantiomeric forms each having a specific chirality. Warner-Lambert also stated that in the '893 patent the disclosure is not limited to compounds having such a specific chirality, and thus, each isomer of the '893 patent is a mixture of enantiomers and not the currently claimed individual enantiomers having an R chirality.

114. In response, the PTO examiner again rejected Warner-Lambert's argument that the original Lipitor patent did not anticipate the R-trans enantiomer and, on November 7, 1990, issued a final rejection on anticipation grounds. The examiner determined that the '893 patent described the R-trans enantiomer of atorvastatin and pointed out that Warner-Lambert's arguments were carefully considered, but not persuasive. Where a reference discloses a genus or

compound of similar structure which are sufficiently limited in number, the reference is deemed to provide description of those compounds just as specifically as if they were identified by name. The examiner further observed that to isolate the claimed invention from the compounds disclosed in the '893 patent, one merely has to select from the limited possibility of isomers to arrive at the claimed invention, and separate them using conventional techniques.

4. The renewed application: Warner-Lambert submits the Roth declaration, again falsely claiming the R-Trans enantiomer is ten times more active than the racemate

115. On February 29, 1991, Warner-Lambert filed a request for retroactive extension of time for revival of the application, a preliminary amendment, and a declaration by Dr. Roth ("Roth declaration"). The declaration, which was submitted to overcome potential rejection based on obviousness, again claims a "surprising" and "unexpected" tenfold increase in activity. It falsely professes to present seemingly objective evidence of an unexpected characteristic of the isolated R-trans enantiomer, and Warner-Lambert claimed this characteristic would allow issuance of an R-trans enantiomer patent despite the claimed invention being *prima facie* obvious in light of the '893 patent. The Roth Declaration simply presented more of the same: affirmatively false and misleading biological data.

a. Warner-Lambert admits the R-Trans enantiomer is *prima facie* obvious

116. While Warner-Lambert presented technical reasons as to why the proposed R-trans enantiomer patent was not "anticipated" by the original Lipitor patent, Warner-Lambert also raised, on its own, the issue of obviousness. Indeed, Warner-Lambert admitted that the R-trans enantiomer was *prima facie* obvious in light of the '893 patent. In its remarks in support of the renewed patent application, Warner-Lambert directed the examiners' attention to a decision of the U. S. Court of Customs and Patent Appeals, *In re May and Eddy,* 197 USPQ 601, 607

(1978), which states: "As recognized in *In re Williams,* 36 CCPA 756, 171 F. 2d 319, 80 USPQ 150 (1948), the novelty of an optical isomer is not negated by the prior art disclosure of its racemate."[13] Warner-Lambert asserted that this case law clearly applied to its patent application.

117.

118. In May, the applicant *conceded prima facie obviousness*, but submitted "rebuttal evidence" in the form of four declarations that it was "unexpected" that the compounds in question did not exhibit the addictive qualities of most opiates. The PTO refused to consider the rebuttal evidence but the U. S. Court of Customs and Patent Appeals overturned the refusal and made its own findings as to whether (i) the record supports non-addictiveness and (ii) non-addictiveness would have been unexpected to one of ordinary skill in the art. "[B]alancing the *prima facie* case of obviousness made out by the PTO against appellants' objective evidence of nonobviousness," the Court concluded, "the subject matter of claims 11-13 would not have been obvious to one of ordinary skill in the art." *Id.* at 611. *May,* therefore, stands for the proposition that when a claimed invention is *prima facie* obvious, an applicant may provide declarations identifying objective evidence of a surprising characteristic to overcome an obviousness rejection.

For the enantiomer application, Warner-Lambert purported to do just that. In its remarks, Warner-Lambert stated that, in light of the *Williams* case, Warner-Lambert provided by a declaration a comparison among each enantiomer and mixture of enantiomers. This comparison was provided to overcome the Roth reference [that is, the reference in the Original Lipitor '893 patent] of the present rejection to facilitate a finding of patentability and move the prosecution

[13] The facts here are quite distinct from *Williams*. In *Williams*, as here, the applicant sought a patent on a particular enantiomer. The *Williams* court determined that the racemic compound had been disclosed in the prior art, but (in contrast to this situation) the fact that the compound was racemic had not been disclosed prior to the priority date.

toward resolution of pertinent issues. In other words, Warner-Lambert noted, *although the Examiner did not include a rejection under 35 U. S. C. 103 [for obviousness], Warner-Lambert included a rebuttal of such rejection to comply with the Williams case law.*

Warner-Lambert further described the declaration as providing the data as set out in the present application in a manner to provide patentability to the application,[14] and stated that *the declaration was submitted to provide evidence of patentability* to the instant invention.

b. The Roth declaration is affirmatively false and misleading

119. Warner-Lambert submitted the Roth declaration in an effort to overcome the otherwise inevitable rejection on obviousness grounds. The Roth declaration states "the antihypercholesterolemia properties of ["R-enantiomer," or "Compound I"] and ["S-enantiomer," or "Compound II"] and mixtures thereof are assessed using essentially the CSI screen that is disclosed in [the '893 patent]." The declaration continues, claiming that the R-trans enantiomer has "activity greater than *fifty-fold more* than that of Compound II and which indicates activity *at least ten-fold more* than that of the racemate," and contains the following table:

[14] Warner-Lambert thus at least tacitly acknowledges that the CSI table previously submitted in the patent specification is not sufficient to provide patentability.

8. THAT, in said assessment, the datum from the Compound I, the datum from its enantiomer the Compound II and the datum from the racemate of the two compounds I and II are as follows:

	Compound	IC_{50} (micromoles/liter)
I	[R-(R*R*)] isomer	0.025
II	[S-(R*R*)] isomer	>1.00
	Racemate	0.26

9. THAT, the data demonstrate that the Compound I provides an IC_{50} which indicates activity greater than fifty-fold more than that of Compound II and which indicates activity at least ten-fold more than that of the racemate;

120. The Roth declaration gives the impression that all appropriate, reasonably available information regarding CSI assay data is represented in the declaration when it describes "the datum from the compound I" and "the datum from the racemate" of that compound. The declaration further claims that "the differences in the data . . . among Compounds I, II and racemate shows the activity of Compound I is *surprising and unexpected* because if the Compound II is accepted as inactive, the activity of the Compound I would be expected to be only twice that of the racemic mixture."[15] The declaration affirmatively and falsely states that the data "indicates activity at least ten-fold more than that of the racemate."

121. The Roth declaration, like the earlier CSI table, purports to present reliable scientific data but does not disclose the source of that data.[16] Given this lack of specification, a skilled addressee would conclude that Warner-Lambert would not have included the data in

[15] Roth's declaration concludes with a paragraph stating, in part, "these statements are made with the knowledge that willful false statements and the like so made are punishable by fine or imprisonment, or both…and that such willful false statements may jeopardize the validity of the above identified US patent application ... or any patent issuing thereon."

[16] The renewed patent specification also contains a chart (the "CSI chart") showing ten times greater activity of the R-trans enantiomer than the corresponding racemate. The information contained in this chart is identical to that presented in the original application.

paragraph 8 of the declaration in such an unqualified way unless the data had been confirmed by a number of repeat assays. In fact, the Roth declaration presents unreliable data from a single, deeply flawed screen — CSI 118. The declaration is false and misleading.

Figure 9: CSI 118 data

CSI#	Date	Racemic Lactone	R-trans Lactone	S-trans Lactone	Racemic Sodium Salt	R-trans Sodium Salt	S-trans Sodium Salt	Racemic Calcium Salt	R-trans Calcium Salt	S-Trans Calcium Salt
118*	10/24/88				.00977			.257	.0251	> 1.0
					.00913			.234	.0216	

* = test calculated multiple values using different methods

Blue = Roth used in CSI table (discussed *supra*)

Yellow = Roth reported in the Roth Declaration

122. CSI 118 used all three forms of calcium salt in a single head-to-head assay. There is no indication that it was ever re-run to confirm its outcome.[17] The test results are unusable for a number of reasons.

123. First, in order to obtain accurate IC_{50} values, the concentration of the test solutions must be known prior to testing; but Warner-Lambert did not determine the concentration of its test solutions prior to conducting the CSI 118 test. Without accurate information about the concentration of the solutions used in the CSI 118 test, the IC_{50} values obtained in CSI 118 cannot be used to demonstrate a tenfold increase in activity of the R-trans enantiomer over the racemate.

124. Second, Warner-Lambert's own lab books show that the compounds in CSI 118 did not dissolve completely in the stock solution. Using non-homogeneous suspensions can

[17] Roth has admitted that he did not conduct any additional tests to confirm that the biological data presented in the patent was in fact correct: "it is true that [the biological data that was included in the patent] went out without any subsequent tests being asked for by me to repeat that data." *Ranbaxy Australia*, 2006 FCA 1787 at 250.

result in variations in the concentrations of the compound in the assay solution leading to wide variation in the results obtained. Given this limitation, the most that the CSI 118 results can be said to determine is whether a compound has *any* activity, not whether a compound has a twofold, threefold, or tenfold increase in activity over another compound.

125. Third, an acceptable CSI test should record similar results for the racemic sodium salt and the racemic calcium salt. (Roth has agreed that, in general, the results for the racemic sodium salt and the racemic calcium salt should be equivalent or similar.) Yet, in CSI 118, the results of the racemic sodium salt and racemic calcium salt are vastly different, showing as much as a twenty-five-fold difference. The difference was so great that the IC_{50} value for the R-trans enantiomer calcium salt showed far less potency than the racemic sodium salt — that is, the R-trans enantiomer of the calcium salt was less active than the racemate of the sodium salt. This difference should have been a red flag that something was wrong with the screen, likely a problem related to the difficulty with solubility of the compounds.

126. Finally, the claim in the Roth declaration of ten-fold greater activity is also affirmatively false because the activity of the isolated R-trans enantiomer is not in fact ten times greater than the racemate. Had Warner-Lambert employed an acceptable scientific testing process, the data would have revealed the R-trans enantiomer had at best a twofold advantage over the racemate, an advantage that would have been expected, not "unexpected" or "surprising."

127. Roth and Warner-Lambert were aware of the numerous problems with CSI 118 identified above and knew that the results of CSI 118 were not scientifically sound. Yet, in the face of radically different values for the sodium and calcium salts, solubility problems, unknown solution concentrations, and results that showed the racemate of one salt was more potent than

the R-trans enantiomer of another salt, they used this questionable and unreliable data to support the false claim that the isolated R-trans enantiomer has ten times greater inhibition of cholesterol synthesis than the racemate, and specifically claimed this as "a surprising level of activity" which, in turn, supported patentability. Warner-Lambert and Roth admit this: Dr. Roth has admitted under oath that he submitted CSI data for the purpose of demonstrating "a surprising level of activity" which therefore supported patentability:

> Q. So [the biological data] was put in to demonstrate this surprising level of activity for the purpose of obtaining a patent, was it not?
>
> A. [Dr. Roth:] Yes, I guess you would say that that would be true. I mean, the data supported a surprising level of activity, which we thought would be novel and surprising and therefore would support patentability.[18]

128. Warner-Lambert knew that the PTO would read the Roth declaration as fairly reflecting all of the appropriate CSI data available to Pfizer for the relevant compounds, and that the data as a whole provided reasonable grounds for the findings set forth in the Roth declaration. Roth and Warner-Lambert intended that the Roth declaration should be read as suggesting a tenfold increase in activity and therefore supporting patentability.

5. The final rejection: The PTO determines the R-Trans enantiomer is anticipated

129. On September 16, 1991, the PTO examiner issued a final rejection of the follow-on patent application, rejecting all claims under 35 U. S. C. § 102(b) as being anticipated by the '893 patent for the reasons set forth in the two rejections issued in 1990.

[18] *Ranbaxy Australia*, 2006 FCA 1787 at 250.

6. The appeal: the PTO determines the R-Trans enantiomer is *prima facie* obvious

130. On January 15, 1992, Warner-Lambert appealed the examiner's rejection to the Board of Appeals, claiming the R isomer as claimed appeared to be at least *100 times more active than its corresponding S isomer and more than 10 times more active than the mixture* and that, under ordinary circumstances, one would have expected only a two-fold difference between the particular R isomer and the mixture. Warner-Lambert went further, stating that the invention described the particular R isomer which was found to have *greater than 10 times the activity* of the compound described in the prior art reference, namely, the racemic mixture, and that the compound of the invention did not produce substantially the same result since it had *greater than 10 times the activity* than the reference compound. Warner Lambert also stated that the R isomer is the most desired and the most *surprisingly active* isomer of the two possibilities if one is to select from the trans compounds.

131. Acknowledging that the isolated R-trans enantiomer is *prima facie* obvious over the '893 patent, Warner-Lambert argued that the obviousness is overcome by the surprising and unexpected activity claimed in the Roth declaration. Warner Lambert stated that the examiner's rejection was erroneous as a matter of law by applying the facts of the present case to the wrong law, that the issue was whether an optical isomer is novel over its prior disclosed racemic mixture, and that the law as stated in *May* and *Eddy* affirming *In re Williams* says the answer is yes.

132. On March 24, 1992, the examiner filed an answer to Warner-Lambert's appeal. The examiner alleged no new grounds for denial of the application, but reiterated the previously disclosed grounds, stating that even if a preferred isomer were not disclosed by the '893 patent,

one skilled in the art expects one of the individual isomers to be more active than the other since this, too, is knowledge contemporary in the art.

133. On October 19, 1992, the Board of Appeals overturned the Examiner's rejection *for anticipation* of the application, concluding that the '893 patent,

at best, only describes the trans racemate containing the R-trans and the S-trans isomers in admixture. Nowhere does the '893 patent state or suggest which optical isomer is preferred and, moreover, does not specifically mention how one skilled in the art could make the pure optical isomer separately. In light of these observations, the Board of Appeals was unable to subscribe to the examiner's contention that the '893 patent anticipated the claimed subject matter.

134. However, the Board recommended to the examiner that upon remand the patent should be rejected on the basis of *obviousness*.

Upon further prosecution of this application before the examiner, it recommended that the examiner analyze the claimed subject matter under the provisions of § 103 of 35 USC, stating that *an obviousness rejection of claims directed to an optically pure isomer appeared to be in order because, (1) the product of the prior art is known to be racemic and (2) methods for resolving the racemic mixture into the pure optically active isomers are known to those skilled in the art.*

7. The '995 patent issues: PTO relies on biological data to overcome obviousness

135. On March 16, 1993, apparently without any further formal proceedings or briefing, the PTO issued a Notice of Allowability for the follow-on, isolated R-trans enantiomer patent application. The '995 patent issued on December 28, 1993.

136. Warner-Lambert had presented the results of CSI screens in both the ’995 patent specification and the Roth declaration to support the contention that the R-trans enantiomer is surprisingly and unexpectedly *ten times more active than the racemate* and therefore not obvious in light of the ’893 patent. Warner-Lambert made this representation in the original application, the Roth declaration, the appeal to the PTO, and in the final patent specification. This is the only “surprising” activity of the isolated R-trans enantiomer that is ever discussed in the ’995 patent application, and the sole reason Warner-Lambert overcame an obviousness rejection.

137. The PTO relied on the Roth declaration and the CSI table to find that the R-trans enantiomer was not obvious in light of the ’893 patent. The Board of Appeals explicitly (i) directed the examiner to re-evaluate the application for obviousness, and (ii) stated that an obviousness rejection appeared to be appropriate. The examiner did precisely that. The examiner relied on Warner-Lambert’s claim of “surprising” and “unexpected” activity and determined that the charts presented in support of that claim (both in the patent specification itself and the Roth declaration) were sufficient to overcome a rejection on obviousness grounds. The only “surprising” or “unexpected” characteristic of the isolated R-trans enantiomer Warner-Lambert claimed was the tenfold increase in activity compared to the racemic mixture. The only data presented in support of those claims were contained in the patent specification (the CSI table) and Roth declaration.

138. The inclusion of particular language and data in the patent specification itself confirms that the PTO relied on both the claim of surprising and unexpected activity and the data submitted in support of that claim. The specification states, “[i]t is now unexpectedly found that the enantiomer having the R form of [a] ring-opened acid [described in the ’893 patent]… that is [R-(R*R*)]-2-(4-fluorophenyl)- β,δ -dihydroxy-5-(1-methylethyl)-3-phenyl-4-

[phenylamino)carbonyl]-1H-pyrrole-1-heptanoic acid, provides surprising inhibition of the biosynthesis of cholesterol." The specification further states "an ordinarily skilled artisan may not predict the unexpected and surprising inhibition of cholesterol biosynthesis of the present invention in view of [prior] disclosures."

139. But for Warner-Lambert's fraud, the '995 patent would never have issued.

D. 1989-93: Warner-Lambert intended to deceive the PTO

140. Warner-Lambert's false claims and data were made and provided to the PTO with the specific intent that the PTO rely on those claims in order to issue a follow-on patent, and with knowledge they were false and misleading. Roth and Warner-Lambert knew that the PTO would read the CSI table and the Roth declaration as a representation that the results in the table fairly reflected all of the scientifically reliable CSI data available to Warner-Lambert for the relevant compounds, and that the data as a whole provided reasonable grounds for the findings set forth in the CSI table and the Roth declaration. Roth and Warner-Lambert intended that the CSI table and the Roth declaration should be read as suggesting a ten-fold increase in activity and therefore supporting patentability.

1. Warner-Lambert manipulated the existing biological data to show a ten-fold increase in activity and Pfizer; intentionally presented false information

141. Warner-Lambert manipulated the existing biologic data in order to show a ten-fold increase in activity. It did so with specific intent to deceive the PTO.

142. Warner-Lambert has acknowledged that head-to-head testing provides the best way to compare quantitative differences in activity, yet it did not present such head-to-head data in support of its claim of ten-fold activity of the R-isomer over then racemate. Instead, Warner-Lambert cherry-picked results from various tests conducted on different days, using different salts, and suffering from various flaws and presented these cooked-up results in the CSI Table

included in the patent specification. Such an extreme departure from accepted chemistry practice — by a company fully aware of what accepted chemistry practice would have required — shows knowledge of falsity and specific intent to deceive.

143. Warner-Lambert acknowledged that had the results of CSI 107 been included in this "average," there would be no surprising or unexpected result. Warner-Lambert has claimed it did not include CSI 107 because it believed that the compounds it tested were not enantiomerically pure; yet, it included the results of CSI 120, which suffered from a not substantially different level of contamination. Such an extreme departure from accepted chemistry practice shows knowledge of falsity and specific intent to deceive.

144. Warner-Lambert claimed that it did not provide the data from CSI 119 to the PTO because CSI 119 was not a head-to-head comparison, and it claimed it believed that it was inappropriate to compare individual data points from different experiments. Yet, Warner-Lambert used different data points from multiple experiments to generate the data contained in the CSI table. Such an extreme departure from accepted chemistry practice shows knowledge of falsity and specific intent to deceive.

145. Warner-Lambert included one of the three results from CSI 118 in the CSI table in order to show an alleged ten-fold increase in activity. The sodium salt prepared by opening the racemic lactone in CSI 92, 93, 95, and 102 should have given substantially identical, or at least very similar, values to the racemic sodium salt that was separately prepared in CSI 118. Yet, the results for the racemic sodium salt in CSI 118 differ from the results of the four lactone CSI tests by a factor of ten. Such an extreme departure from accepted chemistry practice shows knowledge of falsity and specific intent to deceive.

146. In CSI 118, the results of the racemic sodium salt and racemic calcium salt are vastly different, showing as much as a twenty-five-fold difference. The difference was so great, that the IC_{50} value for the R-trans enantiomer calcium salt showed far less potency than the racemic sodium salt — that is, the R-trans enantiomer of the calcium salt was less active than the racemate of the sodium salt. This difference should have been a red flag that something was wrong with the screen, likely a problem related to the difficulty with solubility of the compounds. Instead, Warner-Lambert used this questionable data to support the false claim that R-trans enantiomer has a tenfold greater inhibition of cholesterol synthesis than the racemate.

147. Warner-Lambert was aware of the numerous problems with CSI 118 identified above and knew that the results of CSI 118 were not scientifically sound. Yet, in the face of radically different values for the sodium and calcium salts, solubility problems, unknown solution concentrations, and results that showed the racemate of one salt was more potent than the R-isomer of another salt, Warner-Lambert used this inconsistent outcome to further substantiate its false claim that the R-isomer was ten times more active than the racemate in inhibiting cholesterol synthesis.

148. Warner-Lambert's patent attorneys submitted the false and misleading CSI table generated by Roth and others and the false and misleading Roth declaration to the PTO in furtherance of a deliberately planned and carefully executed scheme to defraud the PTO to gain approval of the '995 patent application.

2. Warner-Lambert admits the patent specification claims a surprising ten-fold increase in activity

149. At numerous points in the prosecution of the '995 patent, Warner-Lambert and Roth expressly stated that the activity of the R-trans enantiomer was both surprising and ten-times greater than the activity of the racemic mixture. Nonetheless, in subsequent patent

litigation, Roth and Warner-Lambert tried to shy away from admitting that Warner-Lambert had claimed that the surprising feature of the R-trans enantiomer was a tenfold increase in activity over the racemate. Warner-Lambert knew that both the CSI table and Roth declaration presented false information about the activity of the R-trans enantiomer as compared to the S- trans enantiomer and the racemate. To acknowledge in court that the only claimed "surprising" characteristic was in fact false would result in the loss of the '995 patent and/or its foreign counterparts.

150. Roth's evasive testimony on this topic is illustrative:

> Q: I suggest to you that you either do or do not rely on those figures. If you want to put out a merely qualitative statement that you have surprising activity you can put it in words. If you put it out in figures that suggests that it is a very surprising level of activity, being a 10-fold difference?
>
> A: But I believe the words we used were a surprising level of activity. We didn't say that it was surprising because it was a 10-fold difference. We simply said that it was surprising, the numbers suggest 10-fold. But frankly, again, anything
> more than twofold would be surprising. We didn't claim 10-fold in the patent. We said it was surprising.
>
> Q: You didn't put a qualification to the numbers that you give in the patent to say "beware of these numbers. We're only really saying that we get a better than two-fold improvement"; no mention of that, was there?
>
> A: What we say is that the compound has surprising activity and then we put data into the patent which supported the surprising level of activity. I don't think that we actually comment on the data except to say that it's surprising. The data is what the data is.
>
> Q: The data on its face quantify that is surprising level of activity, does it not, Dr. Roth?
>
> A: There are numbers given, yes.
>
> Q: So it quantifies that surprising level of activity?
>
> A: What do you mean by that?
>
> Q: Do you know what the meaning of the word "quantifies"

is?

A: There are numbers that are given. Again, we don't make any claims; all we say is that it's surprising. The numbers are what the numbers are.[19]

151. Roth was ultimately forced to concede that the biological data contained in the patent specification purports to show a ten-fold increase in activity, and that it was included in the specification for that reason:

Q: And you wanted those numbers to be taken at face value, did you not?

A: I'm not sure I know what you mean.

Q: What?

A: The data is what the data is. The data was included to support the rising level of activity. What the numbers suggest is that it's something like 10-fold, but we don't state that. We simply — what we simply do is we say it's surprising.

Q: Isn't it a fair reading of this passage on page 8 that having said it's surprising that you are saying now here is why and you set out figures which show a 10-fold increase and you don't provide any qualification at all to those numbers?

A: That is true. We simply report the data.[20]

152. Roth acknowledged, "[t]he data is what the data is," "the numbers are what the numbers are," and "the data was included to support the surprising level of activity. What the numbers suggest is that it's something like 10-fold[.]" The numbers show, based on cherry-picked test results, regardless of whether particular words appear in the text of the patent, that the R-trans enantiomer is ten times more active than the racemate. In reality, the R-trans enantiomer is, as expected, only about twice as active as the racemate.

[19] *Ranbaxy Australia*, 2006 FCA 1787 at 251.

[20] *Id*.

3. Warner-Lambert intended for the PTO to rely on the false data and claims

153. Roth has admitted under oath that he submitted the selected CSI data for the purpose of supporting a surprising level of activity, which therefore supported patentability: "the biological data that was included in the patent I felt demonstrated and supported a surprising level of biological activity."

> Q. So [the biological data] was put in to demonstrate this surprising level of activity for the purpose of obtaining a patent, was it not?
>
> A. Yes, I guess you would say that that would be true. I mean, the data supported a surprising level of activity, which we thought would be novel and surprising and therefore would support patentability.[21]

E. 1996: The FDA approves Lipitor, and the original Lipitor patent provides years of patent protection

154. On June 17, 1996, Warner-Lambert submitted a new drug application under § 505(b) of the FDCA and 21 C. F. R. § 314. 50, seeking approval to sell atorvastatin calcium. The formulation developed for FDA approval and commercialization was atorvastatin calcium, *i. e.,* the isolated R-trans enantiomer formulated as a calcium salt. On December 17, 1996, the FDA approved atorvastatin calcium — now brand named "Lipitor" — for the treatment of hypercholesterolemia and mixed dislipidemia. The FDA initially approved 10 mg, 20, mg, and 40 mg tablets, adding approval of 80 mg tablets on April 7, 2000.

1. Warner-Lambert lists the '893 and '995 patents in the Orange Book

155. Following approval, under 21 U. S. C. § 355, Warner-Lambert listed both the '893 patent and the fraudulently-obtained '995 patent in the FDA Orange Book. When it did so, Warner-Lambert knew that it had procured the '995 patent by actual fraud on the PTO. By

[21] *Id*. at 250.

listing both patents in the Orange Book, Pfizer forced any generic company seeking approval of an ANDA for generic atorvastatin calcium to file a Paragraph IV certification as to both the '893 and '995 patents if the generic company wished to enter the market prior to the expiration of both patents. Such a certification would, Pfizer knew and intended, trigger the ability of Warner-Lambert to file infringement litigation, which in turn would trigger the usual Hatch-Waxman statutory delays for ANDA approval by the FDA *(i. e.,* the 30-month stay of ANDA approval).

156. At the time of FDA approval of Lipitor, the '893 patent was scheduled to expire on May 30, 2006. The '995 patent, by contrast, was scheduled to expire on December 28, 2010.

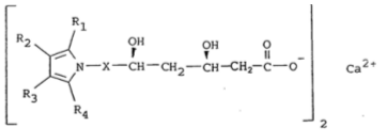
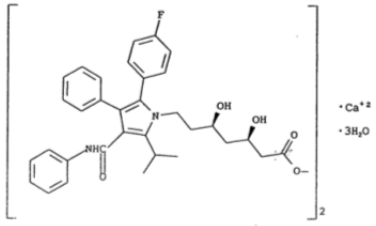
2. The '893 original Lipitor patent protected the Lipitor franchise for years

157. Shortly after FDA approval**,** Warner-Lambert filed with the PTO an application under 35 U. S. C. § 156 for an extension of the term of the '893 patent. Section 156 provides that the period of patent protection may be extended in order to account for the time lag between the issuance of a patent covering the active ingredient in a new drug and FDA approval.

158. Warner-Lambert asked the PTO to extend the period of market exclusivity granted by the '893 patent — not the '995 patent — for about three years and four months. Again, Warner-Lambert took the position that the '893 patent covered the isolated R-trans enantiomer, atorvastatin calcium.

159. Warner-Lambert informed the PTO that (i) the FDA had approved Lipitor, (ii) the active ingredient in Lipitor was atorvastatin calcium, and (iii) atorvastatin calcium is covered by the '893 patent. Warner-Lambert claimed that the '893 patent claimed atorvastatin calcium as a new chemical entity (Claims 1-4), as a pharmaceutical composition (Claim 8), and as a method to inhibit cholesterol biosynthesis (Claim 9).

160. Claim 1 requires "a compound of structural formula I" or "a hydroxyl acid or pharmaceutically acceptable salt thereof, corresponding to the opened lactone ring of the compounds of structural formula I above." In the extension application, Warner-Lambert claimed that Lipitor is a pharmaceutically acceptable salt of structural formula I, and thus covered by Claim 1 of the '893 patent:

> Lipitor is a pharmaceutically acceptable salt (i. e., calcium salt) of the hydroxy acid corresponding to the opened lactone ring of a compound of structural formula I. Lipitor has the general structure:
>
> 
>
> where: X is $-CH_2CH_2-$
>
> R_1 is 4-fluorophenyl;
>
> R_2 is phenyl;
>
> R_3 is -CONH-phenyl; and
>
> R_4 is 1-methylethyl, an alkyl group having three carbon atoms.
>
> Lipitor™ thus has the specific chemical structure
>
> 

161. The PTO granted the patent term extension.

162. Ultimately, with both an extension for the time spent pursuing FDA approval of Lipitor under Section 156, and then for pediatric testing pursuant to the statutory provisions of the FDCA providing for additional marketing exclusivity, the '893 patent would expire on March 24, 2010.

163. Warner-Lambert also sought and obtained a six-month extension for pediatric testing for the '995 patent pursuant to the FDCA. As a result, the expiration date of the '995 patent was June 28, 2011.

164. In effect, the '893 patent would provide more than thirteen years of patent exclusivity to market and sell branded Lipitor — from the 1997 launch until March of 2010. The '995 patent — fraudulently procured by Warner-Lambert from the PTO — would tack on, if enforced by Warner-Lambert or its successors, another 15 months of protection from generic Lipitor competition.

3. In 1997, Warner-Lambert and Pfizer launch Lipitor

165. Prior to commercialization, Warner-Lambert decided it wanted to employ a "saturation" approach to selling Lipitor. The intent of the "saturation" strategy was to have as many sales representatives as possible contacting physicians. As Anthony Wild, Warner-Lambert Pharmaceutical Sector President, explained, "[t]he more soldiers you have out there, the more guns, the more likely you are to achieve your ends." A 1995 sales force deployment study revealed that the Warner-Lambert's sales force was inadequate in size and focus to effectively launch Lipitor.

166. Warner-Lambert therefore chose Pfizer to assist in marketing Lipitor. Warner-Lambert and Pfizer outgunned the competition with the largest sales force ever. Between Warner-Lambert and Pfizer, more than 2,200 sales representatives were believed to be selling Lipitor during its launch in the U. S.

167. After launching in January 1997, Lipitor reached $1 billion in domestic sales within its first 12 months on the market. By the end of 1998, Lipitor was available for sale in 50 countries. In October 1997, 30% of all new statin prescriptions were written for Lipitor.

4. After launch, Warner-Lambert and Pfizer obtained and listed additional patents

168. Subsequent to the 1997 launch for the marketing of Lipitor, Warner-Lambert (and later Pfizer) procured additional patents covering particular (and narrow) processes or formulations ostensibly relating to versions of atorvastatin calcium.

169. First, in November of 1997, Warner-Lambert procured patent 5,686,104 (the "'104 patent," expiry January 19, 2013), and in October of 2000 procured patent 6,126,971 (the "'971 patent," expiry November 11, 2014). Both the '104 and '971 patents cover particular, and narrow, ways of formulating atorvastatin calcium with various excipients to stabilize the finished pharmaceutical product. These two patents are referred to as the "Unasserted Stabilization Formulation Patents;" "unasserted" because despite later efforts by generic companies to enter the market, Pfizer did not assert these two patents against them; "stabilization" because the composition mentioned in the patents contemplates a particular way of achieving stabilization in the final product, and "formulation" because the two patents only cover two narrow formulations of atorvastatin calcium products.

170. Second, in October of 1999, Warner-Lambert procured patent 5,969,156 (the "'156 patent," expiry July 8, 2016). Generally speaking, the '156 patent is for the crystalline form of atorvastatin calcium. (To obtain this patent Warner-Lambert told the PTO that around the end of its safety and efficacy studies in 1995 it had reformulated its atorvastatin calcium from an amorphous to a crystalline form).

171. Third, in July 2000, Warner-Lambert procured patent 6,274,740 (the "'740 patent"). In August of 2001, Warner-Lambert acquired patent 6,087,511 (the "'511 patent"). Both the '740 and '511 patents are process patents, i.e., they cover processes that ostensibly relate to a

manner in which to make atorvastatin calcium. These two patents are called the "Process Patents."

172. Pfizer listed the Unasserted Stabilization Formulation Patents and the '156 patent in the FDA Orange Book as covering Lipitor. As a practical matter, however, Pfizer knew that as narrow process or formulation patents, would-be generic makers could design-around these patents. The Process Patents were not listed in the Orange Book; as patents for a particular process to make a drug, they are ineligible to be listed in the book.

F. 2003: Pfizer files sham litigation against Ranbaxy based on the '995 patent

173. Ranbaxy was the first to file an ANDA for generic Lipitor. On August 19, 2002, Ranbaxy filed ANDA 76-477, seeking approval to sell a generic version of Lipitor in the 10 mg, 20 mg, 40 mg, and 80 mg tablet strengths.

174. As the first to file a substantially-complete ANDA for generic atorvastatin calcium, Ranbaxy was entitled to 180 days of marketing exclusivity under the then-effective provisions of the FDCA. No other ANDA applicant for generic Lipitor could receive FDA approval until the expiration of Ranbaxy's period of marketing exclusivity, which would not commence running until the earlier of either the inception of Ranbaxy's actual commercial marketing or the last court decision finding all patents listed for Lipitor in the Orange Book invalid or not infringed.

175. In or around February of 2003, Ranbaxy sent two Paragraph IV certifications to Pfizer with respect to all patents listed in the FDA Orange Book (i.e., the '893, '995, '156, '971 and '104 patents). In them, Ranbaxy asserted that no valid patent claims covering Lipitor would be infringed by the sale, marketing, or use of Ranbaxy's ANDA product.

176. In response, Pfizer, within the 45-day period provided by the Hatch-Waxman statutory scheme, filed an action in the United States District Court for the District of Delaware, alleging that Ranbaxy's ANDA product would infringe the '893 and '995 patents (the "*Ranbaxy* litigation"). Pfizer did not allege infringement of the Unasserted Stabilization Formulation Patents or the '156 patent.

177. From 2003 to 2006, the Pfizer's infringement litigation against Ranbaxy based upon the '893 and '995 patents progressed through discovery, a trial (in 2004), a district court decision (in 2005), and an eventual appeal and decision by the United States Court of Appeals for the Federal Circuit (in 2006). Two features of the district court Ranbaxy proceedings are noted here.

178. First, in pre-trial proceedings Pfizer attempted to amend its complaint to add new patent infringement claims based on the '511 and '470 Process Patents. However, process patents may not be listed in the FDA Orange Book and, therefore, could not serve as a basis for Pfizer's infringement action against Ranbaxy under the Hatch Waxman Amendments. Accordingly, the district court denied Pfizer's motion because claims under these two Process Patents would be "premature."

179. Second, at the time the district court rendered its decision regarding the '995 patent in December of 2005, neither the district court nor Ranbaxy had the benefit of portions of critical evidence regarding Warner-Lambert's false statements to the PTO. During the Ranbaxy trial, Pfizer and Roth misrepresented several critical features of Warner-Lambert's biological testing of atorvastatin enantiomers and racemates.

180. First, because COR assays had shown only the expected two times relationship, Pfizer's evidence claimed that the COR assay was "not a reliable assay," "certainly not to

develop an assay or clearly not for a [structural] analysis," that you could "draw no conclusions from the COR screen with regard to structure or activity," and that COR assays conducted as late as 1989 continue to suffer the same kinds of methodological problems as had existed back in 1982.

181. Second, because CSI tests showed wildly different results (as a result of methodological problems, thus dispelling the notion that a few selected CSI tests could show the purported ten times relationship), Pfizer's evidence claimed that Warner-Lambert chemistry personnel were completely ignorant of any methodological issues with respect to the preparation of CSI or COR assays from the biology department in Warner-Lambert, and that there were no methodological problems in the CSI assays which would have ever raised even a question regarding the ability to use CSI tests to make quantitative comparisons between compounds.

182. Third, because AICS assays had shown only the expected two times relationship, Pfizer's evidence claimed that *in vivo* AICS tests were not a reliable measure of comparative activity, that Roth "paid very little attention" to it, and that Roth would not use AICS data to make quantitative comparisons between the activities of compounds.

183. Finally, because Warner-Lambert's PTO submission had used only a few cherry-picked data points rather than informed, repeated studies prepared to show some semblance of honest scientific fact, Roth and Pfizer represented that quantitative structural activity research at Warner-Lambert in the late 1980s did not involve the repetition of assay testing nor the use of statistical analysis.

184. The district court relied on these representations in its ruling that found Roth's PTO submissions regarding the alleged ten-fold biologic power of the enantiomer over the racemate were not made with intent to deceive.

185. Each of these fundamental facts -- misrepresented by Pfizer during the *Ranbaxy* trial -- was untrue. Largely on the basis of evidence not introduced at the *Ranbaxy* trial and often acquired since the time of the *Ranbaxy* trial (through trial proceedings in other, foreign jurisdictions), the truth is the opposite.

186. In fact, Roth himself used COR assay data in making quantitative comparisons of the inherent biologic activity of compounds, and specifically used it to discern quantitative differences in compounds to develop the structure of atorvastatin.

187. In fact, methodological issues in conducting CSI tests that would lead to significant inter and intra-assay variability preventing quantitative comparisons of the enantiomer to its racemate were known and significant. The method used by biology to dissolve the atorvastatin-related compounds routinely encountered difficulties. The lactone ring was not consistently and fully opened to enable consistent testing. As a result, the large quantitative disparate results shown between the racemate and the enantiomer simply displayed differences in how the cherry-picked data points resulted from different degrees of dissolution rather than inherent biologic activity. (Pfizer would eventually concede that the CSI tests ought not to be used as a basis to justify the enantiomer patent).

188. In fact, AICS *in vivo* data was used in making quantitative comparisons of the inherent biologic activity of compounds, and specifically used to discern inherent quantitative differences in compounds to develop the structure of atorvastatin.

189. And, in fact, Warner-Lambert in the late 1980s used test conditions that required repeated testing at multiple doses, and the results of those tests were subjected to statistical analyses in order to justify the next steps in quantitative structural activity research.

190. Because the ’995 patent was fraudulently procured by Pfizer, and because Pfizer withheld material facts from the district court, the *Ranbaxy* case as to the ’995 patent was an objectively baseless sham, and was interposed merely to interfere with Ranbaxy’s ability to market generic Lipitor in competition with Pfizer.

191. On November 2, 2006, the Federal Circuit reversed the district court’s ruling, which had upheld the validity of the ’995 patent, determining that claim 6 – the sole claim that Pfizer claimed Ranbaxy’s ANDA product infringed – was technically invalid (essentially for a scrivener’s error). The Federal Circuit refused to address the district court’s other determinations regarding the ‘995 patent. The Federal Circuit did affirm the ruling that the ‘893 patent was valid and would be infringed by Ranbaxy’s product.

192. Based upon the Federal Circuit’s mandate, in late 2006, the district court amended its final judgment order to enjoin the effective date of any approval of Ranbaxy’s ANDA for generic Lipitor until March 24, 2010 (the expiry of the ’893 patent) and to remove from its final judgment order any prohibition of effective FDA approval of Ranbaxy’s ANDA based on the ’995 patent. The district court’s final judgment order, as amended, was sent to the FDA.

G. 2007-2009: Pfizer seeks reissuance of the fraudulently-procured ’995 patent

1. The background of the ‘995 re-issue efforts

193. In the absence of Warner-Lambert’s fraud on the PTO, the ’995 patent would never have issued. Without the ’995 patent being issued in 1993, no reissue proceeding for a ’995 patent in 2007-2009 would have occurred. Without the reissue proceedings, the reissue patent that did emerge from that proceeding, reissue patent RE-40,667 (the “’667 patent”) would not exist. As a result, the PTO’s eventual decision on the reissue proceedings is irrelevant to this antitrust action.

194. The reissue proceedings do, however, confirm what Pfizer had long known: the biologic data submitted as part of the application for the ’995 patent was false, inaccurate, incorrect, and riddled with errors. And by buying off Ranbaxy’s opposition to the reissuance of ‘995 claims, along with a sleight-of-hand with respect to its submissions to the PTO, Pfizer got the PTO to finally allow, albeit incorrectly, several claims of the ‘995 patent as the ‘667 patent.

195. In August of 2006, the Federal Circuit ruled Claim 6 of the ’995 patent invalid; as a dependent claim, Claim 6 ostensibly narrowed Claim 2 by claiming “the hemi-calcium salt of the compound of Claim 2.” However, Claim 2 itself was a dependent claim limited only to atorvastatin acid and did not include salts. Thus, Claim 2 and Claim 6 dealt with “non-overlapping subject matter,” and the claims had been improperly constructed. As a result, the 2006 Federal Circuit ruling held Claim 6 invalid, but the court declined to address any further issues regarding invalidity of the ’995 patent that had been litigated before the district court in *Ranbaxy*.[22]

196. In January 2007 and in the wake of the 2006 Federal Circuit ruling tossing the vital Claim 6 of the ’995 patent on technical grounds, Pfizer sought reissuance of the ’995 from the PTO to correct an alleged technical defect in some of the patent claims. In doing so, Pfizer sought to limit the PTO’s review to a determination whether the newly proposed re-wording of the claims (to correctly construct dependent or independent claims) would satisfy the construction rules of applicable patent laws.

197. While at the outset Pfizer sought only to correct a technical defect, it knew that huge problems lurked behind the scenes for the ’995 patent, and that the PTO or others might

[22] *Pfizer, Inc. v. Ranbaxy Laboratories Ltd.*, 457 F.3d 1284, 1292 (Fed. Cir. 2006).

raise the far more substantive problem that the ’995 patent was simply an obvious extension of the original ’893 patent (and that the data to support a finding of surprising or unexpected activity of the enantiomer was false). By this time of early 2007, the ’995 patent and its nearly identical foreign counterparts had been the subject of considerable litigation, not only in the federal district court *Ranbaxy* proceeding (with its limited in scope appellate review), but also in other countries throughout the world. Through these foreign proceedings, Pfizer learned it could no longer get away with publicly relying upon the falsified biological data to support a claim that the r-trans enantiomer of atorvastatin was ten times stronger than racemic atorvastatin (or indeed that it was anything other than the expected double strength). As a result, Pfizer expressly disavowed to the PTO the reliability of the 1989-1993 biological data as a basis to reissue any of the claims in the ’995 patent.

2. The re-issuance proceedings show that the biologic data could not support a basis to issue the ’995 patent

198. On January 16, 2007, Roth and Pfizer submitted the claim 6 ’995 patent reissue application. The applicants did not amend or modify the ’995 patent specification as part of the reissue proceedings. Roth’s remarks include a list of the “objective evidence” that “completely refutes any suggestion of obviousness.” But now, the list does *not* include the purported surprising effectiveness of the R-trans enantiomer or a purported ten times greater activity of the R-trans enantiomer than the racemate.

199. An Informational Disclosure Statement of the same date states:

> Subsequent to the Federal Circuit’s decision, while preparing for trial in Australia on a ’995 counterpart, Pfizer first learned of *significant errors* in the COR results which neither Pfizer nor the parties adverse to it had discovered before. This discovery led Pfizer to advise the Federal Circuit that COR data could not be relied on to compare the relative activity of compounds — see Exhibit 9, page 10, fn 2. Thus *any earlier reference in Pfizer’s*

> *findings, conclusions and brief to relative activity among compounds based on the COR test is withdrawn and is not relied on in these reissue proceedings. Pfizer does not at this point in the reissue rely for patentability on any comparisons based on CSI.* Neither CSI nor COR data were relied on by either U. S. court in reaching their decisions regarding the validity of '995 claim 6.

Elsewhere Pfizer states, "Pfizer does not now rely on any…data [comparing between and among calcium salts and other salts of atorvastatin and its racemates] in support of patentability."

200. In May 2007, Ranbaxy filed a protest with the PTO against Pfizer's reissue application. Ranbaxy would continue protesting for about another year until, pursuant to a comprehensive agreement to be discussed later, Ranbaxy stopped doing so.

201. On June 7, 2007, as part of reissue proceedings on the '995 patent, Pfizer submitted a Second Informational Disclosure Statement that discusses "Foreign Proceedings on '995 Counterparts" and attached additional materials produced as part of certain non-U.S. proceedings. Pfizer acknowledged that the biological data submitted in support of their patent applications — in the CSI table, the Roth declaration, and the foreign "'995 counterparts" — is inaccurate:

> [A]pplicant is submitting these documents to permit the Examiner to consider their potential materiality. Further, many of these documents . . . contain biological data or summaries of biological data, and *some of that biological data is now understood to be inaccurate* (due to transcription errors, calculation errors, experimental errors, etc.).

202. Elsewhere in the reissue proceedings, Pfizer referred to the biological data at issue in the Australian and Canadian patent litigation as "biologic data that Pfizer *then* argued showed that the atorvastatin enantiomer had unexpected and surprising inhibition of cholesterol biosynthesis in-vitro in comparison to the racemic form of atorvastatin," while reiterating that

they "*are not relying on any of the biological data as a basis for the patentability of the pending claims at the present time*." Similarly, Roth and Pfizer stated, "[a]pplicant is not submitting *corrected* biological data at the present time because, as applicant has emphasized repeatedly in these reissue proceedings, applicant is not currently relying on the biological data for patentability."

203. At one point in the reissue proceedings, the examiner, seeing Roth's late 1980s representations in the PTO record, relied on the biological data to overcome an obviousness rejection:

> Claims 6, 13 and 14 have not been rejected as being obvious as the declaration of Bruce D. Roth filed February 25, 1991 discloses unexpected properties which would overcome any 35 USC 103(a) rejection of claims 6, 13 and 14 as atorvastatin calcium was shown to have activity greater than fifty-fold more than that of the S-trans and at least ten-fold more than that of the racemate.

204. Pfizer knew it could no longer allow the PTO to use its falsified biological data. As a result, it "reiterated [to the PTO] that they are not presently relying on any of the biological data (including the data contained in the Roth declaration) as support for the patentability of claims 6, 13 and 14." It stated:

> Although applicant believes that the evidence provided in the Roth Declaration is sound, and is in no way disclaiming this data, it does not believe that it is necessary to consider such evidence in view of the present record . . . applicant respectfully requests that the Examiner withdraw her reliance on the data in the Roth Declaration and focus instead on the overwhelming evidence of secondary considerations that are discussed above…

The referenced secondary considerations include the argument based on Lipitor's commercial success.

205. On August 2007, the PTO issued a First Office Action rejecting Pfizer's reissue application on grounds set forth in Ranbaxy's May 2007 protest – that certain claims in '995 patent were anticipated, obvious, or constituted double-patenting.

206. On April 24, 2008, the PTO issued a non-final rejection of claims 6, 13, and 14. In so doing, the examiner stated, "[a]s the data contained in the Roth declaration has not been relied on by Applicant in the instant reissue and is not a comparison of the claimed subject matter (atorvastatin calcium) to the closest prior art, the examiner withdraws the reliance on the data in the Roth Declaration to overcome an obviousness rejection of reissue claims 6, 13 and 14."

3. No legitimate basis existed to reissue the claims in the '995 patent

207. Recognizing that the original basis for issuing the '995 patent was false and fraudulent, Pfizer manufactured new reasons why, on the basis of information available 15 years after the '995 patent was issued, the '995 should still re-issue despite its obviousness over the original '893 Lipitor compound patent.

208. Pfizer should have addressed the pertinent question: whether the '995 R-trans enantiomer patent was obvious given the coverage for atorvastatin already in the original '893 compound patent. For an enantiomer patent to overcome an objection of obviousness in light of its parent compound patent, the enantiomer must have some surprising and unexpected attributes beyond those of the compound.

209. Instead of focusing on the pertinent question, Pfizer's reissue application repeatedly characterized the question before the PTO as whether "Lipitor" had experienced commercial success warranting, as a secondary consideration, a conclusion that it was non-obvious. Pfizer's 2007 reissue application and its later support read more like promotional

pieces for Lipitor to sell the PTO on Lipitor's marketing success, rather than have the PTO focus on the actual issues to be decided by the PTO.

210. But Pfizer knew that this argument of generally looking at "Lipitor" (rather than distinguishing attributes of the enantiomer that were surprising and unexpected) was a deception Pfizer knew that Lipitor was protected by the '893 patent from the initial launch of Lipitor through all of the re-issue proceedings. Thus, any showing of success of Lipitor generally would not in any way elucidate why the '995 patent (which *also* covered Lipitor) was not obvious over the original '893 compound patent. Indeed, Warner-Lambert, and later Pfizer, repeatedly identified the '893 patent as the patent which would provide protection for Lipitor. Warner-Lambert listed the '893 patent in the Orange Book, thus protecting Lipitor from generic competition.[23] Shortly after Lipitor was approved by the FDA in late 1996, Warner-Lambert sought, and obtained, a patent extension on the '893 patent (not the '995 patent) to make up for many years that it took to study Lipitor. And Pfizer later brought infringement cases against generic companies arguing that their proposed Lipitor products would infringe the '893 patent.

211. Put simply, from late 1996 to 2009, Pfizer's commercialization of Lipitor was actively protected by both the original '893 Lipitor compound patent and the '995 patent, i.e., both patents covered the commercialized R-trans enantiomer calcium salt formulation. Thus, any arguments raised with the PTO at any time regarding the commercial success of "Lipitor" could not, as a matter of fact or law, elucidate in any way whatsoever whether or not the '995 patent was non-obvious over the '893 patent.[24]

[23] The use code used for the '893 patent to cover Lipitor was a "method of inhibiting cholesterol biosynthesis in a patient." Similarly, the use code used for the '995 patent was defined as a "method of use to inhibit cholesterol synthesis in a human suffering from hypercholesterolemia."

[24] Notably, Pfizer's re-issue application stated that the re-wording of the '995 patent should be allowed so that the "active ingredient responsible for Lipitor's success [could] be restored and the active ingredient that makes Lipitor

212. Outside of the '995 proceedings, Pfizer has admitted that commercial success of Lipitor cannot be used as a basis to distinguish between the '893 and '995 patents. According to Pfizer it would not be appropriate to infer the non-obviousness of *two* unrelated patents based on the success of a *single* commercial product.

213. By April of 2008, the PTO, with Ranbaxy as an objector, had repeatedly rejected Pfizer's reissuance efforts.

214. In June of 2008, however, Pfizer and Ranbaxy entered into an agreement (later described in this complaint as the Delay Agreement). Under that agreement, Ranbaxy either expressly or impliedly agreed to discontinue it protests to the pending reissuance proceedings.

215. With Ranbaxy out of its way, Pfizer continued to barrage the PTO with information about the commercial success of "Lipitor," treating it as if that were the correct and only relevant issue.

216. Eventually, the PTO relented to Pfizer's barrage of Lipitor materials regarding commercial success.

217. On April 6, 2009, the PTO reissued claims 6, 13, and 14 of the '995 patent as the '667 patent. The PTO based its ruling to grant the re-issuance of the '995 patent not on the basis of the biological studies and representations made by Warner-Lambert (even though a version of the CSI assay data remains in the specification for the patent), but instead on the basis of Pfizer's

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work will again be protected by species claims," falsely suggesting that without the allowance Lipitor would be without patent protection. This was a false suggestion because Lipitor's active ingredient was also covered by the original '893 patent as well. Similarly, Pfizer's reissue application misleadingly referred only to the portion of the 1993 ruling of the board of appeals decision which held the '995 patent not anticipated by the '893 patent; Pfizer completely ignored the portion of that same ruling which determined that an enantiomer patent under circumstances such as this case would be obvious over an original compound patent. Elsewhere, Pfizer stated that "one molecule - the molecule specifically claimed in Claim 6 of this re-issue application is responsible for the success."

arguments that the commercial success of Lipitor shows that the '995 patent could not have been obvious.

218. Were it not for Pfizer's fraud on the PTO in the context of procuring the '995 patent, there would never have been a '995 patent in the first place, nor any commercial success attributable to the '995 patent for the patent examiner to rely upon to reissue the '995 patent.

H. Pfizer Files a sham "citizen petition" with the FDA

219. During the 30 month period from early 2003 until about August of 2005, the *Ranbaxy* case had stayed final FDA approval of Ranbaxy's generic Lipitor ANDA.

220. As August of 2005 approached, Ranbaxy's ANDA was the only pending ANDA on file for generic Lipitor, and Pfizer knew that after the end of the 30-month stay FDA could issue final approval for Ranbaxy's generic Lipitor ANDA (pending since August 2002), which in turn would permit generic Lipitor competition to begin. Pfizer also knew that as a matter of procedure and practice, FDA did not issue tentative approvals to ANDA filers as to whom the applicable 30-month stay had expired; it issued final approvals only. So Pfizer would have no warning of when the ANDA approval might occur.

221. Pfizer wanted to delay such final approval for as long as it could.

222. As a result, beginning in July of 2005, Pfizer sent a series of communications, including a "citizen petition," to the FDA. Both the timing and content of these submissions were a sham, sent not for a proper purpose but as an attempt to slow down the FDA approval process for Ranbaxy's ANDA.

223. To demonstrate how the content and timing of Pfizer's letter and petition were a sham, some background is required.

1. The FDA had a long-standing policy that drug substances having differences in polymorphic forms were not considered different active ingredients for ANDA purposes

224. Salts of atorvastatin are polymorphic. The polymorphs can be either crystalline or amorphous. Crystalline forms have different arrangements and/or conformations of the molecules in the crystal lattice. Amorphous forms consist of disordered arrangements of molecules that do not possess a distinguishable crystal lattice. The FDA has a long history of established policies as to how to address polymorphs in the ANDA review and bioequivalence context.

225. In 1992 (15 years before Pfizer made its submissions to the FDA), the FDA specifically rejected a regulatory proposal that would have required an ANDA applicant to show that the active ingredient (i.e., the drug substance) in its generic drug product and the active ingredient (i.e., the drug substance) in the corresponding brand drug exhibit the same physical and chemical characteristics, that no additional residues or impurities can result from the different manufacture or synthesis process, and that the stereochemistry characteristics and solid state forms of the drug have not been altered (the "1992 regulatory rejection"). By 1992, the FDA had already determined that differences in drug substance polymorphic forms, including difference in residues and impurities, do not cause drug substances to be considered different active ingredients for the purposes of ANDA approvals within the meaning of the FDCA and FDA regulations.

226. This position was reaffirmed ten years later in 2002 when the FDA declined to utilize special or additional scrutiny or specifications when reviewing ANDAs for drug products that use different polymorphic forms of the active pharmaceutical ingredient. On February 15, 2002, in a publicly-available denial of another company's citizen petition of which Pfizer had

actual and/or constructive knowledge (the "Ceftin decision"), FDA stated that "FDA's view is that the [FDCA], existing regulations, preamble statements, and the FDA publication *Approved Drug Products With Therapeutic Equivalence Evaluations (Orange Book)* [already] provide an adequate basis to guide the Agency's decision-making on ANDAs seeking approval of a generic drug product whose active ingredient has a different physical form than the active ingredient in the reference listed drug."[25] (Italics in original.) The generic ANDA filer in the Ceftin decision was, as here, Ranbaxy.

227. In its rejection of the Ceftin petition, the FDA reiterated that "FDA's review of any ANDA [already] includes ensuring that the ANDA applicant has the appropriate controls in place with respect to the drug substance and drug product. In FDA's view, Ranbaxy has appropriate controls with respect to the drug substance and the drug product."[26] Thus, FDA expressly declined to apply special or additional scrutiny or specifications to the review of an ANDA when a different polymorphic form of the active pharmaceutical ingredient was used by the proposed ANDA product.

228. The FDA explained that under existing FDA standards what mattered in connection with ANDA approval was the performance of the *drug product* (not the active ingredient (i. e., the *drug substance* in isolation)):

> If a polymorph displays different properties such as melting point, solubility, and stability, these characteristics could ultimately have an impact on the approval of an ANDA for a proposed generic *drug product*. These characteristics could ultimately affect the approval because the approval is based not only on whether the active ingredient in the proposed generic drug product is the "same" as the active ingredient in the reference listed drug, but also on whether the proposed generic *drug product* is the same as the reference listed drug. FDA will

[25] 2/15/2002 Letter from D. Baker to D. Beers, D. Korn, W. McNichol, M. Scheineson, and T. Frisch.

[26] *Id.*

> approve a generic drug product if the ANDA applicant provides, among other things, sufficient information to show that the generic *drug product* is the "same" as the reference listed drug. However, if the active ingredient of a proposed generic drug product were to have a different polymorphic form than the active ingredient in the reference listed drug, and this difference affected the behavior or certain characteristics of the drug product, then FDA might not approve the generic drug product, despite the fact that the proposed generic drug product contained the same active ingredient as the reference listed drug.[27]

229. The FDA also announced in the Ceftin decision that:

a. "[a] difference in the physical form of an active ingredient in a generic drug product from the physical form of the active ingredient in the reference listed drug, including a difference in the crystalline structure of the active ingredient, does not bar the approval of a proposed generic drug product;

b. a generic drug product to be regarded as having the same active ingredient under [21 C. F. R] § 314. 92(a)(1), the drug substance in a proposed generic drug product need not have the same physical form as the drug substance in the reference listed drug; and

c. FDA's scientific expertise and experience have shown that a difference in the physical form of the active ingredient in a generic drug product from the physical form of the active ingredient in the reference listed drug, including a difference in the crystalline structure of the active ingredient, does not prevent a finding of therapeutic equivalence."[28]

230. In 2003, prominent scientists within FDA's Center for Drug Evaluation and Research publicly stated that there was no scientific basis upon which to conclude that an ANDA

[27] *Id.*

[28] *Id.*

applicant's using a different drug substance polymorph, compared with the corresponding brand drug, would preclude the ANDA applicant from demonstrating drug product manufacturability, bioequivalence, and stability. Those same FDA scientists also stated, at or around the same time, that there was no scientific or regulatory basis for requiring a generic drug product to use the same polymorphic form as the innovator.

231. Those same FDA scientists also stated, at or around that same time, that despite the potential effect that polymorphism may have on drug stability, *because drug product stability is affected by a multitude of other factors, including formulation, manufacturing process, and packaging, it is the stability of the drug product, not the drug substance, that is the most relevant measure of drug quality*. Thus, existing FDA scrutiny of ANDAs was sufficient when polymorphic forms of drug substances were involved, according to FDA. According to the FDA scientists, under cGMPs, the sponsor of the ANDA must still provide evidence of manufacturing process validation and demonstrate that the drug product can be manufactured reproducibly, while meeting all the required in-process, release, and stability specifications.

232. Moreover, in a December 2004 draft guidance (the "2004 Polymorph Draft Guidance"), FDA explained that polymorphism was already accounted for by existing FDA regulations and ANDA review procedures:

> In addition to meeting the standards for identity, each ANDA applicant is required to demonstrate that, among other things, the drug product exhibits sufficient stability and is bioequivalent to the [corresponding brand drug]. While the polymorphic form can affect drug product stability and bioequivalence, these performance characteristics are also dependent on the formulation, the manufacturing process, and other physicochemical properties *(e. g.,* particle size, moisture) of both the drug substance and formulation excipients. Using a drug substance polymorphic form that is different from that of the [corresponding brand drug] may not preclude an ANDA applicant from formulating a generic drug product that exhibits bioequivalence and stability, and *the drug substance in the*

> *generic drug product need not have the same polymorphic form as the drug substance in the [corresponding brand drug].*[29]

233. FDA reiterated in the 2004 Polymorph Draft Guidance what the FDA scientists had said in 2003**: "**because drug product stability is affected by a multitude of other factors, including formulation, manufacturing process, and packaging, *it is the stability of the drug product, and not stability of the drug substance polymorphic form that should be the most relevant measure of drug quality."*[30]

234. Although given the opportunity to comment upon the 2004 Polymorph Draft Guidance, Pfizer did not do so.

235. At all relevant times, the preface to the FDA "Orange Book" provided that "[a]nhydrous and hydrated entities, as well as different polymorphs, are considered pharmaceutical equivalents[.]"

236. Any reasonable drug company, and certainly Pfizer as the largest drug company of all, would know FDA's established policy and practices regarding polymorphic forms of active pharmaceutical ingredients prior to sending their letter and petition to the FDA.

2. Pfizer's own amorphous form of atorvastatin was known to present fewer safety risks than the crystalline form

237. Pfizer also knew, from its own work with atorvastatin, that the amorphous form of atorvastatin actually presented *fewer* concerns for the safety of patients than the crystalline form.

238. According to Pfizer's statements to the FDA, virtually all of the clinical studies conducted in the early 1990s to support the safety and efficacy of atorvastatin in humans had used batches of the amorphous formulation of atorvastatin. However, around 1995 – late in the

[29] Guidance for Industry, ANDAs: Pharmaceutical Solid Polymorphism, U.S. Dep't of Health and Human Services (December 2004).

[30] *Id.*

clinical development and the FDA approval processes – Pfizer decided (of its own accord, and not due to any FDA concerns) to switch to the crystalline form of atorvastatin for the product it would ultimately market.

239. With one exception, all of Pfizer's preclinical studies were done using the amorphous form of atorvastatin. But one 13-week comparative studies in rats, mice, and dogs done in July 1995 used the crystalline form and provided Pfizer with cause for concern – there was mortality among dogs receiving the crystalline form after only 3 months of treatment, while no mortality among dogs given the amorphous form. Moreover, the locations and numerous sites of impact from the crystalline form were troublesome.

240. Pfizer's late shift from an amorphous to a crystalline form of atorvastatin was a major concern to FDA reviewers overseeing the Lipitor NDA – not because the amorphous formulation posed safety concerns, but because the change to the crystalline version did. Pharmacokinetic studies showed that the crystalline version had an early, and high, spike in its dissolution profile, meaning that a greater amount of atorvastatin was present in the blood much faster than with the amorphous form; this spiking, it was surmised, might lead to some of the higher safety risks for the higher doses seen in clinical studies for the crystalline formulation.

241. Eventually during the NDA process, Pfizer persuaded FDA to approve all but the highest dose (80 mg) of the crystalline form of atorvastatin (the 80 mg was taken off the table at the time due to crystalline concerns). But the process made clear that in comparisons between amorphous versus crystalline atorvastatin, the amorphous form presented fewer concerns for patient safety than that of the crystalline form.

3. Pfizer knew that Ranbaxy's proposed ANDA product used the amorphous form of atorvastatin, not the crystalline form used in Lipitor

242. Finally, Pfizer knew from the *Ranbaxy* litigation that Ranbaxy's ANDA proposed that its generic Lipitor use amorphous atorvastatin calcium as its active pharmaceutical ingredient.

4. Pfizer seeks sham relief and files a sham citizen petition with FDA

243. In July of 2005, Pfizer sent a letter entitled "Generic Versions of Atorvastatin" to the FDA. In it, Pfizer said it was "concerned" that ANDA applicants for generic Lipitor were using amorphous atorvastatin calcium, which, Pfizer claimed, "may be susceptible to higher levels of impurities than are found in Lipitor and that may degrade more quickly and thus have inferior stability compared to Lipitor." Pfizer said that this "may raise questions about the approval of" ANDAs for generic Lipitor. Pfizer asked FDA to "carefully scrutinize" such "potential differences in quality . . . before the atorvastatin variants are approved under ANDAs." Pfizer said that "the risk of reduced quality in the generic product," due to the use of amorphous atorvastatin, was "clear," and that Ranbaxy's ANDA should be "reviewed with considerable skepticism."

244. The letter to FDA was signed by a Pfizer scientist, a vendor Pfizer used that "provides analytical services to the pharmaceutical industry," and was copied to Jeffrey B. Chasnow, a lawyer in Pfizer's legal department.

245. Pfizer's true purpose for sending the letter to the FDA was to cause the FDA to take a longer time in reviewing Ranbaxy's ANDA for generic Lipitor and thereby delay final approval once the 30-month stay had ended.

246. On August 30, 2005, the FDA informed Pfizer that the procedure for communicating with the FDA on such issues was to file them as a so-called "citizen petition."

247. On November 7, 2005, Pfizer re-filed the July 28 letter as a citizen petition. Pfizer restated its request: "Pfizer asks that FDA consider the information provided in the July 28 letter, together with any additional information that may be submitted to the petition file by Pfizer or others, in FDA's decisions concerning approvals of generic versions of atorvastatin."

248. Having studied the amorphous form thoroughly, Pfizer knew—and told the FDA in or around June of 1995—that there were no clinical safety or efficacy implications related to using the amorphous, as compared with the crystalline, form. As it turned out, toxicity was a concern for the *crystalline* form of atorvastatin calcium that Pfizer ultimately used, not the amorphous form that Pfizer abandoned (and Ranbaxy proposed to use).

249. Pfizer submitted no evidence to the FDA that showed or even suggested that Ranbaxy's ANDA product, because it used amorphous atorvastatin calcium as the drug substance (i) would not be pharmaceutically equivalent or bioequivalent to branded Lipitor, (ii) would not demonstrate satisfaction of the conditions for approval under the FDCA, or (iii) would not be capable of being processed or manufactured under current good manufacturing practices ("cGMP"). Instead, Pfizer simply ignored the FDA's prior stated positions concerning polymorphism, and submitted its petition in contradiction to these principles.

250. Pfizer's letter and petition were objectively baseless and interposed solely to create an obstacle to the final FDA approval of Ranbaxy's generic Lipitor ANDA. No objectively reasonable petitioner would have expected success on the merits of Pfizer's letter or petition. Both lacked any reasonable regulatory, scientific, medical, or other reasonable basis. Pfizer's letter and petition lacked any evidence that lent support to their assertions or that bore on the approvability of Ranbaxy's ANDA product. Neither stood any chance of affecting the FDA policy or procedure. Both were flatly contrary to the FDA's expressed views regarding drug

substance polymorphic forms, and did not reasonably argue (or argue at all) for a change in those expressed views. In short, Pfizer's letter and petition were nothing more than a thinly-veiled effort to impose delay and meaningless regulatory effort upon the FDA to document the existence and application of accepted regulatory processes on the FDA's review and approval of Ranbaxy's final ANDA approval.

251. Of course, Pfizer's timing in making these submissions to the FDA was also a sham. In July of 2005, Pfizer had no more information than it had had for many months, if not years, earlier about Ranbaxy's ANDA; it purposely submitted the letter to the FDA shortly before the 30-month stay was to expire, in an attempt to obtain additional delay as a result of the submission of that letter – delay which, Pfizer hoped, would continue after the 30 month stay expired.

5. The FDA denies Pfizer's sham petition

252. On November 30, 2011 – the same date that under an agreement (to be discussed later) between Pfizer and Ranbaxy, Ranbaxy could first enter the market with generic Lipitor – the FDA issued its formal written denial of the Pfizer petition.

253. The FDA denied Pfizer's petition because, just as it had already said in the 1992 regulatory rejection, the 2002 petition response, the 2004 Polymorph Draft Guidance, and repeatedly thereafter, ANDA applicants need not show that their active ingredients have no additional residues, impurities, or solid state forms relative to the active ingredient in the corresponding brand drug.

254. Likewise, as the FDA had repeatedly said before, the FDA's existing policies and procedures were adequate to identify any ANDA product which used different polymorphs than the corresponding brand product, determine whether that difference resulted in any differences in

measures such as purity or stability, and if such differences existed, whether the purity and stability data for the ANDA product satisfied the FDA's longstanding standards for such measures. There was nothing about this process that required any additional skepticism or special consideration by the FDA. Thus, the FDA again expressly declined to apply special or additional scrutiny or specifications to the review of such ANDAs:

> *We believe that the Agency's existing recommendations to industry on assessing active ingredient sameness and stability of polymorphic forms of drug substances*, as well as those on comprehensive chemistry, manufacturing, and controls (CMC) and impurities, are adequate to enable an ANDA applicant to address any potential drug product stability, degradation, and impurity issues associated with the amorphous form of atorvastatin. *We also believe that the Agency's existing policies and review practices are sufficient for a critical evaluation of the variables that have the potential to affect drug product quality of drug products containing amorphous atorvastatin.*
>
> * * *
>
> In the preamble to the final rule implementing the generic drug approval provisions of the Hatch Waxman Amendments, FDA specifically rejected the suggestion that the Agency adopt a requirement that active ingredients "exhibit the same physical and chemical characteristics, that no additional residues or impurities can result from the different manufacture or synthesis process; and that the stereochemistry characteristics and solid state forms of the drug have not been altered."

255. Moreover, in denying the Pfizer's petition, FDA again stated (as it had in the 2004 Draft Polymorph Guidance) that "the inherent stability of the *drug substance* polymorphic form should not be the primary consideration in making a determination of *product* stability. Rather, the stability of the generic atorvastatin drug *product* is the most relevant measure of drug product quality." (Emphasis in original.)

256. Again, these were not new positions on the part of FDA. Instead, Pfizer's petition was flatly contrary to, and willfully ignored, FDA's previous decisions and previously-expressed

views in the 1992 Regulatory Rejection, the 2002 Decision, the 2004 Polymorph Draft Guidance, and repeatedly thereafter. Pfizer had no objectively reasonable basis to file the letter or petition.

I. In 2008, Pfizer shifted from unilateral to conspiratorial unlawful conduct in order to preclude generic entry

257. By 2008, a markedly changed landscape afforded Pfizer no practicable opportunities to unilaterally extend its marketing exclusivity for Lipitor beyond March of 2010. After all, (i) the ’995 patent would expire in March of 2010, (ii) Claim 6 of the ’995 patent (the only asserted claim against Ranbaxy) was adjudicated invalid, (iii) its effort to gain re-issuance of the ’995 had been met with protests by Ranbaxy and rejections by the PTO, (iv) its two process patents could not apply to Ranbaxy’s product (and would likely not apply to other generic efforts), (v) its two stabilization formulation patents had never been, and could never be, asserted against Ranbaxy (and likely any other generic company), and (vi) the petition it had filed with FDA lacked all merit and would, in time, be rejected.

258. To set the stage for the allegations of Pfizer’s *conspiratorial* unlawful activities in the face of this dilemma, the complaint first sets out in more detail these circumstances.

1. The ’893 and ’995 patents could only bar generic entry until March of 2010

259. As to the ’893 patent, the *Ranbaxy* district court’s entry of final judgment in the end of 2006 barred generic entry by Ranbaxy until March of 2010. As to the ’995 patent, however, the final order adjudged claim 6 (the only ’995 claim asserted against Ranbaxy) invalid (in accordance with the Federal Circuit’s ruling regarding the scrivener’s error); as a result, claim 6 of the ’995 patent could not, as a matter of law, be enforced against Ranbaxy. As a result, when in early 2008 Pfizer looked at its strategic options, it could only expect the ’893 and’995 patents to bar generic entry by Ranbaxy until March of 2010.

2. There was no valid basis for re-issuance of the ’995 patent

260. In early 2008 things looked bleak, as they should have, for the ‘995 reissuance. In April of 2008 the PTO had rejected the application; since Pfizer was no longer relying upon the biological data, there was no basis to conclude the claims in the ‘995 were anything other than obvious over the ’893 patent. Ranbaxy’s protests were being heard by the PTO. And no logical basis existed to otherwise grant the reissuance request.

3. The two Unasserted Stabilization Formulation Patents and the ’156 patent had never been, and could never be, validly asserted against Ranbaxy

261. In 2008 and when assessing strategic options, Pfizer could gain no solace from its two Unasserted Stabilization Formulation patents, nor the ’156 patent, as methods to preclude Ranbaxy entry.

262. As to the two Formulation Patents (i.e., the ’971 and ’104 patents), neither patent had yet been used as the basis for an infringement action against Ranbaxy, nor could they. Both patents were for narrow formulations to achieve stabilization for particular atorvastatin drug products, and thus did not apply to Ranbaxy’s proposed product under its ANDA.

263. As to the ’156 patent, it covered *crystalline* forms of atorvastatin, not amorphous ones. But Ranbaxy’s product was the latter. Pfizer did not and could not show that Ranbaxy’s product would infringe the Formulation Patents or the ’156 patent, and as of today, Ranbaxy’s product is presumed not to infringe these patents.

4. The two Process Patents would not apply to Ranbaxy’s (nor likely other generic) products

264. The two Process Patents also did not provide a vehicle to delay generic entry of Ranbaxy’s (or indeed most if not all other) would-be generic makers.

265. The ’511 and ’740 patents have applications that trace back to a common application and therefore the specifications for both are virtually identical. The Summary of the Invention sections of these two patents are identical and, states:

> [T]he present invention is a novel process for the preparation of amorphous atorvastatin and hydrates thereof which *comprises* . . . (a) dissolving *crystalline Form I atorvastatin* in a non-hydroxylic solvent; and (b) removing the solvent to afford amorphous atorvastatin”

(emphases added).

266. The Process Patents are narrow in scope. For a generic manufacturer’s process to infringe either of these patents, the generic manufacturer must, *inter alia*, start by dissolving *crystalline Form I atorvastatin* in the specified solvent. If the manufacturing process dissolves any crystalline structure other than Form I in the specified solvent, or dissolves amorphous atorvastatin, the process does not and cannot infringe either of the Process Patents. The process must also meet each of the other claims of the Process Patents, as well.

267. Because of the narrow scope of the Process Patents, and the ample number of both amorphous and crystalline forms of atorvastatin that were available, a very large number of non-infringing alternatives existed to the technology claimed in the Process Patents. Indeed, the prior art, including the ’893 patent (covering the active ingredient of Lipitor, atorvastatin calcium), describes numerous processes for making atorvastatin calcium that are prior art to the Process Patents and would invalidate the claims of the Process Patents if those claims read on the processes described in the ’893 patent.

268. During Pfizer’s own development of Lipitor, Pfizer first produced (for years) amorphous atorvastatin in their manufacturing processes before (much later) developing crystalline formulations such as Form I. There is no need for someone seeking to produce amorphous atorvastatin calcium to first produce Form I crystalline atorvastatin calcium.

269. Pfizer knew that generic companies would design around process or formulation patents such as these. It is common practice for experienced generic companies such as Ranbaxy to conduct patent searches during the drug development process, and to select drugs and approaches to formulating products that are allegedly covered by patents that the generic companies can readily design around.

270. Process patents cannot be listed in the FDA's Orange Book, because they are not patents claiming an approved drug or an approved use of a drug. The existence of the Process Patents did not, therefore, provide a vehicle for immediate patent litigation, nor did it create a regulatory impediment to generic entry. (ANDA filers are not required to file Paragraph IV certifications with respect to non-listed patents; as a result, Pfizer could not obtain an automatic 30-month stay of FDA approval of an ANDA by bringing a timely suit for infringement of the Process Patents).

271. Nor did the existence of the Process Patents create any significant design or legal impediment to generic entry, even when litigation might be ripe. Numerous non-infringing alternatives to the processes claimed in the Process Patents existed such that there was no reasonable likelihood that Pfizer would be able to use the Process Patents to obtain a court order enjoining ANDA filers, including Ranbaxy, from selling generic versions of Lipitor on the ground that they infringed the Process Patents.

5. Pfizer created the illusion of litigation to create the appearance of patent life beyond March of 2010

272. Agreements between branded and generic companies can often create a vehicle to extend unlawfully branded market exclusivity. With a pending court case Pfizer could settle with Ranbaxy, it could share its Lipitor market exclusivity and associated monopoly profits with

Ranbaxy, extend existing market exclusivity, and later try to argue its settlement was lawful. But in early 2008 Pfizer had no litigation against Ranbaxy that it could settle.

273. So in order to eventually craft agreements to resolve litigation ostensibly relating to patents that extended Lipitor protection beyond March of 2010, Pfizer needed to first create the illusion of litigation that involved patents that applied to Lipitor and were being asserted against Ranbaxy. And since Pfizer wished to extend further the purported patent life for Lipitor, it needed the illusion of litigation involving patents with a life beyond March 24, 2010 (the date the original '893 patent would expire) and beyond June 28, 2011 (the date on which the fraudulently-obtained '995 patent would expire, assuming Pfizer could somehow apply it to Ranbaxy). Pfizer turned to the two Process Patents, the '740 and '511 patents -- covering the reverse processes (from crystalline back to amorphous) for making amorphous atorvastatin calcium.

274. Despite the fact that the *Ranbaxy* court had previously rejected Pfizer's attempt to include the Process Patents in the *Ranbaxy* litigation due to a lack of standing, and despite the fact that no reasonable litigant would expect success on the merits, Pfizer decided that it would bring suit against Ranbaxy on the two process patents.

275. On or about March 24, 2008, Pfizer filed a complaint in the United States District Court for the District of Delaware alleging that Ranbaxy infringed the Process Patents ("*Ranbaxy II*"). Thus, nearly five years after it first attempted to sue Ranbaxy for infringing the Process Patents, and knowing that a court had already ruled that it lacked standing under 28 U.S.C. §§ 2201 and 2208 to do so, Pfizer again sued Ranbaxy for declaratory judgment of infringement of the very same Process Patents on the very same grounds that earlier resulted in dismissal.

276. A lawsuit based on the Process Patents was not justiciable years earlier. It was less so in *Ranbaxy II*. At the conclusion of the *Ranbaxy* case, the final judgment permanently enjoined Ranbaxy from engaging in the manufacture, use, offer to sell or sale of its generic version of Lipitor until the expiration of the '893 patent (March 24, 2010). Thus, in *Ranbaxy II* Ranbaxy itself argued "any harm to Pfizer from alleged infringement of the [Process Patents is] much less imminent now than in the [*Ranbaxy*] case when the Court found no imminent threat of harm or injury." There was no jurisdiction for *Ranbaxy II*. Pfizer knew this.[31]

277. The Ranbaxy II complaint contained only the most conclusory of infringement allegations. The complaint included no factual allegations or support establishing that Ranbaxy's process satisfied the various elements of the claims of the Process Patents. The complaint did not even allege that Ranbaxy starts with the crystalline atorvastatin when making amorphous atorvastatin. Instead, it merely concludes:

> 30. Upon information and belief, Ranbaxy's Atorvastatin Product is made or is intended to be made by a process which if practiced in the United States would infringe the '511 patent.
>
> * * *
>
> 41. Upon information and belief, Ranbaxy's Atorvastatin Product is made or is intended to be made by a process which if practiced in the United States would infringe the '740 patent.

278. These allegations were completely baseless as a matter of fact and law. There was no basis for Pfizer to believe that Ranbaxy was unaware of the elements of the Process Patents. Nor was there any basis to believe that Ranbaxy did not develop a manufacturing process that purposely avoided infringing on those patents. There were numerous forms of

[31] Likewise, because process patents cannot be listed in the Orange Book, Pfizer could not (and did not) use the Process Patents to obtain the automatic 30-month stay of FDA approval of a pending ANDA.

atorvastatin, other than the crystalline Form I specified in the Process Patents, that Ranbaxy could have (and, upon information and belief, did) use at the start of its manufacturing process. And, there was no need for Ranbaxy to first create a crystalline form of atorvastatin calcium before forming an amorphous form of atorvastatin calcium.

279. Moreover, Pfizer knew Ranbaxy intended to use amorphous atorvastatin as a starting material in manufacturing another generic atorvastatin drug, Caduet (a combination of Lipitor and another drug, Norvasc). Pfizer had no basis to believe that Ranbaxy would not similarly use non-infringing amorphous atorvastatin, or another non-infringing process to achieve amorphous atorvastatin, in its manufacturing. Nor did Pfizer have any reasonable expectation that during the discovery process it would learn information supporting a claim of infringement of the two Process Patents.

280. During the pendency of *Ranbaxy II*, Pfizer never produced any evidence to support its purely conclusory allegations that Ranbaxy infringed the Process Patents. Nor could it, since such allegations were false and baseless as a factual (and legal) matter.

281. As a result, the Process Patents had no exclusionary power vis-à-vis potential generic competitors, including Ranbaxy. Pfizer did not (and could not) prove the facts necessary to meet its burden of establishing infringement of each element of the Process Patents. Therefore, even though the Process Patents were presumed to be valid and enforceable, they had no exclusionary power. Pfizer could not use the Process Patents to exclude Ranbaxy (or likely any would-be generic entrant) from the market using the Process Patents.

J. 2008: Pfizer and Ranbaxy enter into an illegal horizontal market allocation agreement

1. With the illusion of litigation, Pfizer and Ranbaxy plan a settlement.

282. In the spring of 2008, Pfizer had created the illusion of patent infringement litigation between it and Ranbaxy. This was the only pending litigation between them relating to Lipitor sales in the United States.

283. Pfizer had no realistic likelihood of meeting its burden of establishing that Ranbaxy infringed these Process Patents. Ranbaxy knew this too. No objectively reasonable litigant would have believed otherwise.

284. Pfizer and Ranbaxy also knew that Ranbaxy was the first filer of an ANDA for generic Lipitor. They understood Ranbaxy could market its generic Lipitor for 180 days free from competition from other ANDA filers. And they knew Ranbaxy's 180-day exclusivity gave it the ability – simply by refraining from launching its own generic Lipitor or from relinquishing the right to its 180-day exclusivity period – to prevent (or "bottleneck") other generic competitors from entering the United States market.

285. Consequently, all Pfizer needed to do to delay all generic Lipitor competition was to enter into an agreement with Ranbaxy under which Ranbaxy would agree to delay the launch of its (the first) generic version of Lipitor. As the defendants knew, such an agreement would create a nearly insurmountable obstacle, a bottleneck, to generic competition for all ANDA filers for the duration of any such agreement.

286. So in the spring of 2008, Pfizer and Ranbaxy discussed the terms of an agreement to delay generic entry, in large part under the guise of the illusory *Ranbaxy II* litigation.

2. The illegal horizontal market allocation agreement

287. On June 17, 2008, Pfizer and Ranbaxy executed an agreement to delay generic entry (the "Delay Agreement"). The agreement is an unlawful "pay-for-delay" agreement.

288. To disguise the Delay Agreement's true anticompetitive purpose, Pfizer and Ranbaxy characterized the agreement as, in part, settling the *Ranbaxy II* litigation. That was a pretext for its true anticompetitive goals and accomplishments.

289. The Delay Agreement is an unlawful contract, combination and conspiracy to allocate the entire United States market for atorvastatin calcium to Pfizer until November 30, 2011.

290. Pursuant to the Agreement, Ranbaxy agreed that it would neither (a) compete directly with Pfizer with a generic Lipitor in the United States market nor (b) selectively waive or relinquish its first-to-file 180-day marketing exclusivity so as to permit any other ANDA filer to compete directly against Pfizer with a generic Lipitor in the United States market until November 30, 2011. Absent the Delay Agreement and given the enormous profit opportunity generic Lipitor presented, Ranbaxy would have been highly motivated to pursue either of these courses far earlier. On the basis of all fact and FDA's desire to permit ANDA filers to bring low-cost generic products to market as soon as possible, absent the agreement Ranbaxy would have entered the market for atorvastatin calcium much sooner than it eventually did.

291. In exchange for Ranbaxy's agreement to delay its launch (and not to authorize another ANDA filer to launch) generic Lipitor until November 30, 2011, Pfizer gave Ranbaxy substantial consideration, including forgiveness of outstanding money judgments unrelated to generic Lipitor in the United States and the right to market generic Lipitor in at least eleven foreign markets. Absent Pfizer's compensation to Ranbaxy, Ranbaxy would not have agreed to any amount of delay in its launch of generic Lipitor (or to any delay its authorizing another ANDA filer to launch). At a minimum,

absent the compensation to Ranbaxy, Ranbaxy would not have agreed to delay its launch (or to delay its authorizing another ANDA filer to launch) for as long as it did, and would instead have agreed only to a substantially shorter period of time before which it would enter.

292. The Delay Agreement also ostensibly gave Ranbaxy protection from infringement liability in connection with the variety of patents that purportedly covered atorvastatin. However, that "consideration" was a sham, illusory, and merely inserted into the agreement to disguise the illegal horizontal agreement to allocate the entire United States market for atorvastatin calcium.

293. In fact and at law, no reasonable litigant would realistically expect any existing Pfizer patent to put Ranbaxy (or likely any other relevant ANDA filer) in danger of liability for infringement of any legitimately obtained patent past March of 2010 (the expiry of the '893 patent). No legitimately obtained patent posed any objectively reasonable or realistic threat of infringement liability to Ranbaxy (or likely any other relevant ANDA filer) for making or selling generic Lipitor, other than the '893 patent. And as of the date of the Delay Agreement, the only means by which Pfizer could have prevented a launch by Ranbaxy of generic Lipitor on or after March 24, 2010 (or June 28, 2011) was by obtaining an injunction. But as Pfizer knew in 2008, obtaining such an injunction would have been impossible, because it would have required a showing that Pfizer was likely to succeed on the merits of baseless patent infringement claims.

294. Thus, upon the expiration of the '893 patent, Ranbaxy would have been able to enter with its generic product notwithstanding any later-expiring patent held by Pfizer. Ranbaxy would have done so immediately upon FDA approval. Ranbaxy has expressed its willingness to enter at risk with a generic product of other blockbuster drugs, telling one court that Ranbaxy presently intends to manufacture, use, sell and offer to sell drug products for which the ANDA

has been submitted once the FDA approves the ANDA -- in other words, Ranbaxy would launch its generic once the FDA approved it and would not need to await final resolution of the patent case.

295. An infringement case against Ranbaxy (or any other ANDA filer), based upon any legitimately obtained Lipitor patent that expired after March 24, 2010, would have been (and was, with respect to, for example, Pfizer's suit claiming infringement of Pfizer's Process Patents) an objectively baseless sham. (And even if it is assumed, contrary to fact, that, the '995 patent was obtained legitimately, it expired by June 28, 2011, five months before November 30, 2011.) As a result, the Delay Agreement gave Pfizer protection from generic Lipitor competition beyond the lawful limits of its exclusionary power under any Lipitor-related patent. Nor did Pfizer or Ranbaxy subjectively believe there was any such legitimate threat of infringement from such patents.

296. Pfizer has acknowledged the lack of lawful exclusionary power for a significant portion of this time, *viz.*, from June of 2011, when the fraudulently procured '995 patent was set to expire. In 2005, before the Delay Agreement existed, Pfizer's former Chairman and CEO stated that

there were dozens of generic drug manufacturing companies with a red circle around June 28, 2011. That was the day Pfizer's patent for the anti- cholesterol medication Lipitor was due to expire. Shortly thereafter a number of generic alternatives to Lipitor would be introduced and consumers would have a choice of generic tablets containing atorvastatin calcium.

297. Of course, only the fraudulently-procured '995 patent expired in June of 2011. Other patents purportedly covering Lipitor -- namely the Unasserted Formulation Patents, the '156 patent, and the Process Patents -- would expire between 2013 and 2017. If the Unasserted

Formulation Patents, the Process Patents, and/or the '156 patent had any hope of legitimately keeping generics off the market, Pfizer's CEO would not have ignored them and the literally tens of billions of dollars they would have conferred on his company. His statement that June 28, 2011 is the key date only makes sense if one recognizes — as Pfizer did — that the Unasserted Formulation Patents, the Process Patents, and the '156 patent could not block generics from entering.

3. The operation of the Delay Agreement

298. Nevertheless, pursuant to the Delay Agreement, Ranbaxy agreed not to sell its generic version of Lipitor in the United States until November 30, 2011 — twenty (20) months after the '893 patent (and any associated marketing exclusivities) was scheduled to expire, and five (5) months after the fraudulently-obtained '995 patent would expire, if in fact it was reissued.

299. The reason that Ranbaxy agreed to keep its generic version of Lipitor off the market until well after the legitimate exclusionary power of Pfizer patents had expired was that Ranbaxy was paid handsomely not to compete with Pfizer. Based upon the limited public disclosures to date regarding the terms of the Delay Agreement, Ranbaxy received at least the following compensation in exchange for its agreement to delay coming to market with its generic version of Lipitor in the United States: (1) permission to sell generic versions of Lipitor in at least eleven foreign markets, including Canada, Belgium, Netherlands, Germany, Sweden, Italy, and Australia; and (2) the forgiveness of monies that Ranbaxy owed Pfizer based on one or more court judgments that Pfizer had obtained on infringement claims unrelated to generic Lipitor in the United States.

300. As part of the Delay Agreement, Ranbaxy also agreed not to challenge the validity of any Lipitor patent, including the ’995 patent, which was then the subject of reissuance proceedings. Pursuant to the agreement, Ranbaxy dropped its challenge to the reissuance of the ’995 patent — a challenge which had been successful prior to the date of the agreement.

301. In April of 2009, after Ranbaxy dropped its challenge, the PTO reissued the ’995 patent as the ’667 patent. The ’667 Patent, like the ’995 patent, would expire (and did expire) on June 28, 2011. Nevertheless, pursuant to the Delay Agreement, which provided Ranbaxy with substantial compensation in exchange for its agreement not to compete, Ranbaxy could not sell its generic version of Lipitor until November 30, 2011, a full five months after the ’667 patent expired.

302. In summary, the Delay Agreement was unlawful for at least the following reasons: (a) it constituted an illegal market allocation agreement, pursuant to which Pfizer paid substantial monies to its competitor, Ranbaxy, in exchange for Ranbaxy’s agreement to allocate the entire United States market for atorvastatin calcium to Pfizer through November 30, 2011; (b) it restricted competition in a manner, and to an extent, that exceeds the exclusionary power and potential of Pfizer’s Lipitor patents; (c) it purported to settle patent infringement litigation that arises from a patent (the ’995 patent) that was procured by fraud upon the PTO; and (d) to the extent it purported to settle patent claims against Ranbaxy for infringement of any Lipitor-related patent extending past March 24, 2010, it was (with respect to the Process Patents), or would have been (with respect to the Unasserted Formulation Patents, the ’156 Patent, the ’995 patent, and ’667 patent), baseless sham litigation that Pfizer and Ranbaxy knew had no realistic chance of prevailing on the merits.

303. There is and was no cognizable, non-pretextual procompetitive justification for the Delay Agreement, and/or for the compensation flowing to Ranbaxy under the agreement. Even if there were some conceivable justification, the Delay Agreement—and the compensation flowing to Ranbaxy under the agreement—was not reasonably necessary to achieve it.

304. Defendants did not need to resort to payments from Pfizer to Ranbaxy in order to resolve their patent litigation. To the contrary, according to FTC analyses, in 2004 and 2005, 90% of agreements between brand and generic manufactures settling patent disputes contained no anticompetitive payment from the brand to the generic manufacturer. Like the parties to such agreements identified by FTC, were it not for the anticompetitive payment from Pfizer to Ranbaxy, if Defendants would have entered into an agreement at all, they would have entered into an agreement providing that Pfizer would not compensate Ranbaxy for delay, and that Ranbaxy would enter far earlier than the Delay Agreement provided.

K. Using the unlawful Delay Agreement, Defendants thwarted other ANDA filers from triggering Ranbaxy's 180-Day marketing exclusivity

305. The Delay Agreement sought to prevent other ANDA filers from launching their own generic versions of Lipitor before Ranbaxy did. Ranbaxy's anticipated 180-day marketing exclusivity as the first filer of a generic Lipitor ANDA would mean that only Ranbaxy's own first commercial marketing of its ANDA product would trigger the 180-day period. Before the expiration of that 180-day period, other generic Lipitor ANDA filers could not market their generic versions of Lipitor.

306. The way that other ANDA filers could trigger Ranbaxy's 180-day exclusivity was by obtaining court decisions that all of the unexpired patents Pfizer listed in the FDA "Orange Book" as claiming Lipitor were invalid or not infringed. If another ANDA filer were to obtain such court decisions, Ranbaxy's 180-day first-to-file marketing exclusivity would commence

running, even if Ranbaxy had not yet begun commercial marketing of its ANDA product by that time, and even if Ranbaxy did not want its exclusivity to commence running.

307. Another way that other ANDA filers could circumvent Ranbaxy's 180-day exclusivity was by convincing FDA to deprive Ranbaxy of a 180-day exclusivity period and approve the ANDAs of other generic companies unimpeded by any 180-day period.

308. These two possibilities were of substantial concern to Pfizer and Ranbaxy in 2008, when they reached the Delay Agreement. Pfizer did not want generic Lipitor competition earlier than the November 30, 2011 date provided in the Delay Agreement, and Ranbaxy did not want any involuntary triggering or forfeiture of its anticipated, and enormously valuable, 180-day first-to-file marketing exclusivity. Such events would frustrate the Delay Agreement, and threaten to diminish or eliminate the value of Pfizer's extended exclusivity and of Ranbaxy's 180-day exclusivity. Pfizer and Ranbaxy shared a keen interest in ensuring that Ranbaxy's 180-day exclusivity commenced as late as possible, and was protected by preventing other ANDA applicants for generic Lipitor from coming to market.

309. To prevent the involuntary triggering of Ranbaxy's 180-day exclusivity prior to November 30, 2011, Pfizer (in furtherance of the Delay Agreement) thwarted the efforts of generic manufacturers to obtain judgments of invalidity and/or non-infringement with respect to the Unasserted Formulation Patents.

310. To effectuate this campaign, Pfizer settled cases prior to judgments on the merits, vigorously opposed the efforts of ANDA applicants to obtain declarations that the Unasserted Formulation Patents were invalid and/or not infringed, and otherwise engaged in a pattern of dilatory conduct designed to forestall, prior to Ranbaxy's agreed-upon November 30, 2011 entry

date, judicial decisions that the Unasserted Formulation Patents were invalid and/or not infringed.

1. Apotex

311. For instance, after it received a Paragraph IV certification in December of 2008 from Apotex, Inc. and Apotex Corporation ("Apotex") as to the '995 patent, the Unasserted Formulation Patents, and the '156 patent, Pfizer sued Apotex for infringement of only the '995 patent. Apotex's answer included counterclaims, pursuant to 21 U. S. C. § 355(j)(5)(C), asserting non-infringement and invalidity of the both the '995 patent (and '667 reissue patent), the Unasserted Formulation Patents, and the '156 patent.

312. As the Apotex trial court recognized: "Apotex's hope is to obtain a decision from this Court that the Unasserted Patents are invalid or are not infringed by Apotex's product, thereby triggering Ranbaxy's exclusivity period. Absent such a court ruling (either in this case or in litigation involving another subsequent ANDA filer), Apotex will not be able to market its generic atorvastatin drug until 180 days after Ranbaxy begins marketing its drug, which, as a result of the settlement agreement between Pfizer and Ranbaxy, will not occur until November 2011 at the earliest."[32]

313. In furtherance of the Delay Agreement, Pfizer sought dismissal of Apotex's counterclaims, arguing that they were nonjusticiable.

314. Although the Apotex court denied Pfizer's motion to dismiss, the motion had its intended effect: it delayed discovery and litigation for well over a year and, combined with subsequent litigation delay tactics surrounding discovery and summary judgment motions,

[32] *Pfizer Inc. v. Apotex Inc.*, 726 F. Supp. 2d 921, 926 (N.D. Ill. 2010).

prevented Apotex from obtaining a judgment of non-infringement and invalidity of the Unasserted Formulation Patents and the '156 patent before November 30, 2011.

2. Mylan

315. On May 1, 2009, Mylan sent Pfizer a letter providing notice of Mylan's ANDA submission and intent to market a generic version of Lipitor, supplying a Paragraph IV certification as to the Unasserted Formulation Patents and the '156 patent, and offering confidential access to certain portions of Mylan's ANDA. By June 15, 2009, Pfizer had filed an action against Mylan alleging infringement of only the '156 patent, and seeking a declaratory judgment of infringement of the Process Patents.

316. Mylan filed a motion for leave to file an amended answer containing counterclaims pertaining to the Unasserted Formulation Patents, to obtain a declaration of noninfringement and/or invalidity with respect to them. In support of that effort, Mylan sought discovery regarding the Unasserted Formulation Patents. Mylan's motion to compel discovery was granted by court order on August 25, 2010.

317. But Pfizer continued to refuse to supply Mylan with the discovery it required. Mylan was forced to file an emergency motion to enforce the court's discovery order.

318. To frustrate Mylan's continued efforts to obtain discovery and thus proceed with its counterclaims pertaining to the Unasserted Formulation Patents, Pfizer, on August 30, 2010, hastily covenanted not to sue Mylan on them, hoping to moot Mylan's continued efforts to discover facts that would assist its counterclaims and the court's order of August 25, 2010 compelling that discovery.

319. The court expressed frustration with Pfizer's litigation tactics regarding the Unasserted Formulation Patents, and enforced its order requiring Pfizer to supply discovery to Mylan pertaining to the Unasserted Patents:

> I'm granting Mylan's request. I'm very troubled by the conduct of Pfizer here with respect to this ongoing discovery dispute. The way I see it, if Pfizer wanted to provide a covenant not to sue, it was within its authority at any time to provide the covenant not to sue with respect to the formulation patents. For whatever reasoning only known to Pfizer, they waited until August 30th [2010] to give the covenants not to sue, which was perhaps not coincidently shortly after the issuance of the August 25th order granting the defendants' request for discovery * * * That's simply just not how this is supposed to work.

320. Pfizer continued to delay the progress of the case. In a November 20, 2010 letter to the court regarding Dr. Reddy's Laboratories Ltd.'s ("DRL") request to be heard at the *Markman* hearing in the Mylan patent litigation pertaining to the '156 patent, counsel for Mylan complained about Pfizer's continued dilatory tactics: "Pfizer uses DRL's request to be heard on the '156 patent as another opportunity to attempt to delay the Pfizer-Mylan cases."

321. Mylan also sought to remove Ranbaxy's blocking 180-day exclusivity period by way of a separate action against FDA seeking an order requiring FDA to determine whether or not Ranbaxy was entitled to a 180-day first-to-file marketing exclusivity.

3. Actavis

322. In August of 2010, Pfizer sued Actavis Group hf, Actavis Inc., Actavis Elizabeth LLC and Actavis Pharma Manufacturing Private Ltd. (collectively "Actavis") after Actavis submitted to FDA an ANDA seeking approval to market generic Lipitor. Although Actavis had included the Unasserted Formulation Patents in its Paragraph IV certification, Pfizer sued Actavis only for infringement of the '156 patent.

323. In September 2010, Actavis counterclaimed for declaratory judgment of invalidity and non-infringement of the Unasserted Formulation Patents. Pfizer moved to dismiss these counterclaims as unripe. In opposing that motion, Actavis argued that "Pfizer's listing of the [Unasserted Formulation Patents] in the Orange Book and its refusal to litigate them creates patent uncertainty and indefinitely delays the approval of Actavis' ANDA," and noted that "[e]ven if Pfizer granted Actavis a covenant not to sue on the [Unasserted Formulation Patents], however, it would not address the fact that Actavis is suffering from an indefinite delay in FDA approval of its ANDA and its concurrent inability to enter the market."

324. Actavis also argued that, by virtue of Pfizer's agreement with Ranbaxy and its refusal to litigate the validity and infringement of its Unasserted Formulation Patents, "Actavis is being restrained from the free exploitation of non-infringing goods, it is suffering exactly the type of injury-in-fact that is sufficient to establish Article III standing" (internal citations and quotations omitted).

325. Despite their efforts to do so, no ANDA filer was able to circumvent the Delay Agreement between Pfizer and Ranbaxy by triggering Ranbaxy's 180-day marketing exclusivity prior to November 30, 2011.

L. Ranbaxy's ANDA would have been approved earlier absent Defendants' anticompetitive scheme

326. Ranbaxy's atorvastatin calcium ANDA would have received final approval earlier absent the defendants' anticompetitive conduct. The FDA has policies and procedures in place to prioritize the review of ANDAs, e.g., expediting the review of the first applications for which there are no blocking patents or exclusivities. Regarding the FDA's review of applications for generic Lipitor, the Delay Agreement blocked the applicants, including Ranbaxy, from marketing their products. The FDA was aware that the earliest date Ranbaxy could market generic Lipitor

under its Agreement with Pfizer was November 30, 2011. As Ranbaxy maintained the 180-day exclusivity, all subsequent applicants were blocked from marketing generic Lipitor as well, until Ranbaxy's exclusivity was triggered and had elapsed.

327. Furthermore, the FDA was under tremendous pressure, including from Congress, to speed consumer access to generic Lipitor at the earliest possible moment. Ranbaxy was also under tremendous pressure to monetize its biggest asset, i.e., its first-to-file ("FTF") atorvastatin ANDA, at the earliest possible moment, so much so that Ranbaxy paid Teva a large amount of money — in effect an insurance policy — in order to ensure that Ranbaxy was able to launch generic Lipitor at the earliest possible moment.

328. As it turned out, Ranbaxy was granted final approval on November 30, 2011, i.e., simultaneously with the earliest possible moment Ranbaxy could market generic Lipitor under the Delay Agreement with Pfizer. Ranbaxy launched generic Lipitor in advance of that date, under "quarantine" agreements with wholesalers. Had the Delay Agreement permitted an earlier entry date, or had there been no such Delay Agreement at all, generic Lipitor could have been, and would have been, marketed earlier than November 30, 2011, because the FDA would have granted final approval earlier and Ranbaxy would have launched earlier.

329. The FDA did not earlier issue its formal written denial of Pfizer' petition until November 30, 2011 for the same reason: the FDA knew from Ranbaxy that the Delay Agreement prevented Ranbaxy from coming onto the market until November 30, 2011. Thus, there was no need for the FDA to issue the formal written denial of Pfizer's Petition earlier than November 30, 2011, and it was for that reason that it did not do so.

1. The longstanding FDA policy of prioritizing the review of ANDAs

330. As a matter of procedure and practice, the FDA has long employed methods of prioritizing the review of pending applications. For example, in 1990 the Division of Generic Drugs within the FDA issued a policy and procedure guide establishing a "first-in, first-reviewed" policy for generic drug applicants. This policy, along with similar guidance for the pharmaceutical industry, has been updated and modified from time to time and is still in place today. One of the modifications which have been instituted over the years is to prioritize the review of the first ANDAs for which there is no blocking patent or exclusivity.

331. Similarly, the FDA has been experiencing a backlog of pending applications, such that prioritizing ANDA review is more important than ever. Furthermore, as a matter of procedure and practice, in a situation where an ANDA filer will not be able to market a drug until a time far into the future, such as Ranbaxy's generic Lipitor ANDA due to the Delay Agreement, the FDA shifts assets to other priorities within the Office of Generic Drugs. FDA prioritizes the review of ANDAs in this way by keeping abreast of the current posture of any litigation that may impact the timing of approval of an ANDA. For instance, as a matter of procedure and practice, upon accepting an ANDA for filing, the FDA expressly requests that the applicant promptly submit a copy of any settlement agreement between the applicant and the patent holder.

2. The FDA's review of Ranbaxy's ANDA for atorvastatin calcium

332. On June 18, 2008 Ranbaxy publicly announced the Delay Agreement in which Ranbaxy's launch date was delayed until November 30, 2011. Ranbaxy submitted this information to the FDA shortly thereafter.

333. Thus, due to the FDA's longstanding policy of prioritizing the review of ANDAs and the recent pressure of the ANDA backlog, on information and belief, once FDA learned of the fact that the first generic for Lipitor, i.e., Ranbaxy, would not be marketed until November 30, 2011, FDA shifted assets away from Ranbaxy's ANDA and the petition and toward other priorities within FDA until the November date drew closer.

3. The tremendous pressure on the FDA to approve generic Lipitor

334. That the FDA was under immense pressure to approve a generic Lipitor product also shows that it would have earlier approved Ranbaxy's ANDA absent the agreed-to date for Ranbaxy's market entry contained in the Delay Agreement.

335. For example, on March 10, 2011 Senate Health, Education, Labor, and Pensions Committee Chairman Tom Harkin, along with Senators Jay Rockefeller, Charles Schumer, Debbie Stabenow, and Sherrod Brown sent a letter to FDA Commissioner Dr. Margaret Hamburg. In the letter the Senators stated, "Given the tremendous savings that access to generic atorvastatin will afford both consumers and the government, we urge you to act now to clarify the relevant regulatory issues in the matter so the public can receive access to a more affordable generic version of Lipitor on the earliest possible date." The "tremendous savings" to consumers and the government would be between "$3.97 billion to $6.7 billion a year upon generic entry, which equates to $10.9 million to $18.3 million a day." Likewise, the FDA recognized the importance and cost savings of having a generic Lipitor available to consumers. [Confirmed]

4. The tremendous pressure on Ranbaxy to market a generic Lipitor and/or otherwise monetize its first-to-file exclusivity

336. Ranbaxy, too, was motivated to monetize its first-to-file 180-day marketing exclusivity, and would have more rapidly pursued its atorvastatin calcium ANDA absent the agreed-to date for Ranbaxy's market entry contained in the Delay Agreement.

337. The first-to-file generic Lipitor was a tremendous opportunity for Ranbaxy. Despite only being on the market with a generic Lipitor for one month of 2011, atorvastatin calcium was Ranbaxy's largest selling product in 2011. Ranbaxy also achieved sales growth of 17% over the previous year, "mainly on account of revenues from First to File product, Atorvastatin, in the US market in December 2011."

338. In order to capitalize on the first to file opportunity, Ranbaxy took steps to ensure issues related its good manufacturing practices did not prevent it from being able to market generic Lipitor. For instance, on information and belief, in December 2009 Ranbaxy effectuated a manufacturing site transfer of atorvastatin calcium from its facility in India to Ranbaxy's wholly-owned subsidiary, Ohm Laboratories in New Jersey. So whatever issues Ranbaxy may have been having with FDA regulatory compliance at one or more of its facilities in India did not affect the Ohm facility in New Jersey. This is borne out by the fact that Ranbaxy ultimately received approval to market generic Lipitor in the U.S. from the Ohm facility in New Jersey.

339. Absent the defendants' anticompetitive scheme, Ranbaxy could and would have proceeded with a manufacturing site transfer earlier, either to Ohm or to another facility. On information and belief, the Ohm facility had been operational for Ranbaxy for quite some time and was available for a site transfer in the relevant time period at issue here.

340. In fact, at or around the same time Ranbaxy filed its ANDA for atorvastatin calcium, Ranbaxy also filed the first ANDA to market a strength of a drug in the same "statin" family as atorvastatin calcium, simvastatin. On information and belief, as with atorvastatin, Ranbaxy effectuated a manufacturing site transfer for simvastatin from India to the Ohm facility in New Jersey. Ranbaxy received final approval for its simvastatin ANDA on June 23, 2006 and began marketing its first-to-file generic shortly after.

341. Similarly, on information and belief, in the same time period as the atorvastatin calcium filing, Ranbaxy filed the first ANDA with FDA to market donepezil hydrochloride. Donepezil hydrochloride is the active ingredient in Aricept. On information and belief, Aricept had approximately $2.6B in sales in 2010. On information and belief, around the time of the atorvastatin calcium site transfer in December 2009, Ranbaxy effectuated a site transfer of donepezil hydrochloride from India to the Ohm facility in New Jersey. On information and belief, on the first day a generic could be marketed, November 26, 2010 Ranbaxy received approval with first-to-file exclusivity to market donepezil hydrochloride. In 2011 donepezil was the second best performing product after atorvastatin calcium.

342. Finally, on information and belief Ranbaxy and Teva entered into an agreement to ensure Ranbaxy was able to market its generic Lipitor at the earliest time that it was ready to do so. Negotiations between Ranbaxy and Teva regarding generic Lipitor began in 2009. Ranbaxy and Teva negotiated three possible ways of monetizing Ranbaxy's first to file ANDA: (1) a manufacturing site transfer from Ranbaxy's facility in India to Teva, under which Teva would pay Ranbaxy a lump sum transfer fee and royalties on sales of generic Lipitor; (2) Ranbaxy and Teva both launch generic Lipitor; and (3) Ranbaxy successfully effectuates the manufacturing site transfer from India to the Ohm facility in New Jersey, pays Teva back the lump sum transfer fee, and Teva shares a portion of Ranbaxy's profits for generic Lipitor. Ranbaxy and Teva signed an agreement regarding generic Lipitor in 2010 containing some or all of these options and/or other options.

343. Once Ranbaxy made the decision to partner with another company in order to monetize generic Lipitor, it is hardly surprising Ranbaxy chose Teva. It is well known in the

industry that Teva looks to partner with 180-day exclusivity holders given the profit opportunity such exclusivities present.

344. Since Ranbaxy gained approval to market generic Lipitor from its Ohm facility in New Jersey, on information and belief, it never needed the insurance policy the deal with Teva effectively provided. However, Ranbaxy still paid Teva a substantial amount of money in order to be able to monetize its first-to-file atorvastatin calcium ANDA at the earliest possible moment under the Delay Agreement.

M. Pfizer takes unprecedented steps to suppress generic substitution

345. In addition to delaying the launch of generic atorvastatin and preventing the commencement (and expiration) of Ranbaxy's exclusivity, Pfizer also took steps to retard the rate at which its monopoly power was dissipated once generic entry had occurred. These steps permitted Pfizer to continue to extract overcharges from Plaintiffs even after the commencement of generic competition.

346. Generic entry typically results in a loss of nearly all of the branded manufacturer's sales as retail pharmacies substitute the less expensive AB-rated generic for the more expensive branded drug. Generic substitution laws and the economic interests of wholesalers, pharmacies, patients and third-party payors typically ensure that, within 90 days of generic entry, 90-95% of prescriptions are filled with the generic rather than the brand. Retail pharmacies and pharmacy chains reap substantial benefits from this substitution process because they purchase generic drugs at lower prices than the corresponding branded drug. The more prescriptions retail pharmacies are able to fill with the generic, the higher their savings. By the same token, any conduct that suppresses the rate of generic substitution causes harm to retail pharmacies by forcing them to buy more of the branded drug and less of the generic.

347. In the months leading up to Ranbaxy's launch, Pfizer took steps to slow down the loss of its monopoly power and delay the benefits of generic competition to Giant Eagle by suppressing the substitution of generic atorvastatin for branded Lipitor. Pfizer accomplished this objective by entering into arrangements with PBMs to give a favored position to branded Lipitor and a disfavored position to generic atorvastatin on their formularies and in their pharmacy benefit plans. Once generic atorvastatin became available, these arrangements raised patient co-pays for generic atorvastatin and lowered patient co-pays for branded Lipitor, with the result that, in many cases, the patient paid a lower co-pay if the pharmacy dispensed Lipitor than if it dispensed generic atorvastatin. In some cases, the arrangement between Pfizer and the PBM ensured that branded Lipitor was covered by the prescription benefit plan and that generic atorvastatin, rather than being covered but disfavored, ***was not covered at all.*** In those cases, the patient would pay only a small co-pay to purchase branded Lipitor but would have to pay the entire retail price of the prescription in order to purchase generic atorvastatin. It is unprecedented for a newly launched generic drug not to be covered by pharmacy benefit plans. As Pfizer was aware, retail pharmacies like the ones owned and operated by Giant Eagle will dispense the brand rather than the generic when it is in the patient's interest to do so, even when it is not in the pharmacy's interest, and the circumstances created by Pfizer ensured that in many cases it was in the patient's interest to stay on the brand. In accordance with its policies, Giant Eagle dispensed the brand in those cases.

348. PBMs and their clients (health insurers, employers and other sponsors of pharmacy benefit plans) generally pay lower reimbursement rates for a prescription filled with a generic than for a prescription filled with a brand. Hence, these entities, like other drug purchasers, typically benefit from generic substitution. In order to overcome the PBMs' normal

preference for generic drugs, Pfizer was required to (and did) promise to pay substantial rebates to the PBMs to induce them to disfavor generic atorvastatin. But while these rebates may have eliminated the harm that PBMs (or their clients) would otherwise have suffered from suppressing the generic substitution rate, they did nothing to eliminate the harm that wholesalers and retailers have suffered in the form of higher acquisition costs.

349. These anticompetitive arrangements between Pfizer and PBMs were scheduled to expire six months after generic entry. That date recently occurred. As a result of these arrangements, the substitution rates for generic atorvastatin at Giant Eagle's pharmacies are substantially below the 90-95% rate that is typical six months after generic entry. Giant Eagle data shows that in June 2012, 87% of the atorvastatin market was genericized. [Stats for July, August, September, October? Those rates are only now starting to move upward and will take some time to reach normal levels. Thus, for six months, and continuing for some time into the future, Giant Eagle paid and will pay more to acquire atorvastatin than it would have paid in the absence of these arrangements. These higher acquisition costs are a direct result of Pfizer's efforts to suppress generic competition and slow down their loss of monopoly power.

350. In addition to being part of Pfizer's unlawful scheme, its efforts to suppress the generic substitution rate after generic entry made economic sense only because of the anticompetitive scheme alleged above, which limited the number of generic entrants and resulted in a relatively high generic price. Absent the anticompetitive conduct alleged above, it is likely that Ranbaxy's 180-day exclusivity would have been triggered and would have expired earlier than it actually did and additional generic entrants would have been on the market during the first six months of generic competition. Under those circumstances, it would not have been

economically feasible for Pfizer to suppress the generic substitution rate of atorvastatin calcium in the manner it did.

VI. INTERSTATE COMMERCE

351. The defendants' efforts to monopolize and restrain competition in the market for atorvastatin calcium have substantially affected interstate and foreign commerce.

352. At all material times, Pfizer manufactured, promoted, distributed, and sold substantial amounts of branded Lipitor in a continuous and uninterrupted flow of commerce across state and national lines and throughout the United States. Beginning around November 30, 2011, Ranbaxy did the same with respect to generic Lipitor.

353. At all material times, Pfizer transmitted funds, contracts, invoices and other forms of business communications and transactions in a continuous and uninterrupted flow of commerce across state and national lines in connection with the sale of branded Lipitor. Beginning around November 30, 2011, Ranbaxy did the same with respect to generic Lipitor.

354. In furtherance of their efforts to monopolize and restrain competition in the market for atorvastatin calcium, the Defendants employed the United States mails and interstate and international telephone lines, as well as means of interstate and international travel. The activities of the Defendants were within the flow of and have substantially affected interstate commerce.

VII. TRADE AND COMMERCE

355. The Defendants' efforts to monopolize and restrain competition in the market for atorvastatin calcium have substantially affected interstate and foreign commerce.

356. At all material times, Pfizer manufactured, promoted, distributed, and sold substantial amounts of branded Lipitor in a continuous and uninterrupted flow of commerce across state and national lines and throughout the United States. Beginning around November 30, 2011, Ranbaxy did the same with respect to generic Lipitor.

357. At all material times, Pfizer transmitted funds, contracts, invoices and other forms of business communications and transactions in a continuous and uninterrupted flow of commerce across state and national lines in connection with the sale of branded Lipitor. Beginning around November 30, 2011, Ranbaxy did the same with respect to generic Lipitor.

358. In furtherance of their efforts to monopolize and restrain competition in the market for atorvastatin calcium, the defendants employed the United States mails and interstate and international telephone lines, as well as means of interstate and international travel. The activities of the defendants were within the flow of and have substantially affected interstate commerce.

VIII. MONOPOLY POWER AND MARKET DEFINITION

359. At all relevant times, Pfizer had monopoly power over atorvastatin calcium because it had the power to maintain the price of atorvastatin calcium at supracompetitive levels without losing substantial sales.

360. A small but significant, non-transitory price increase by Pfizer to Lipitor would not have caused a significant loss of sales.

361. Lipitor does not exhibit significant, positive cross-elasticity of demand with respect to price, with any product other than AB-rated generic versions of Lipitor.

362. Due to the federal and state statutes and regulations governing the marketing of brand and generic drugs, industry practices and Lipitor's use and varying ability to inhibit the production of cholesterol, Lipitor is differentiated from all products other than AB-rated generic versions of Lipitor.

363. Pfizer needed to control only Lipitor and its AB-rated generic equivalents, and no other products, in order to maintain the price of Lipitor profitably at supracompetitive prices.

Only the market entry of a competing, AB-rated generic version of Lipitor would render Pfizer unable to profitably maintain its current prices of Lipitor without losing substantial sales.

364. Pfizer also sold branded Lipitor at prices well in excess of marginal costs, and in excess of the competitive price, and enjoyed high profit margins.

365. Defendants have had, and exercised, the power to exclude generic competition to branded Lipitor.

366. Defendants, at all relevant times, enjoyed high barriers to entry with respect to branded and generic Lipitor.

367. To the extent that Giant Eagle is legally required to prove monopoly power circumstantially by first defining a relevant product market, Giant Eagle alleges that the relevant market is all atorvastatin calcium products — i.e., Lipitor (in all its forms and dosage strengths) and AB-rated bioequivalent atorvastatin calcium products. During the period relevant to this case, Defendants have been able to profitably maintain the price of Lipitor well above competitive levels.

368. The relevant geographic market is the United States and its territories.

369. Pfizer's market share in the relevant market was 100% until the entry of generic atorvastatin in late November 2011.

IX. MARKET EFFECTS

370. Ranbaxy began to ship generic Lipitor on or shortly before November 29, 2011, prior to receiving formal, written final approval of its ANDA from FDA. Ranbaxy informed its customers that such shipments of generic Lipitor were subject to "quarantine," meaning that the generic Lipitor could not be resold until FDA's issuance to Ranbaxy of formal, written ANDA approval.

371. The FDA delayed issuing written approval for Ranbaxy's ANDA until November 30, 2011, because the FDA was informed that the Delay Agreement prevented Ranbaxy from selling generic Lipitor until November 30, 2011. Ranbaxy's ANDA was in an approvable condition well before November 30, 2011 and, were it not for the Delay Agreement, would have received final FDA approval at a much earlier time. By practice, the FDA organizes its priorities around "rate limiters," and the Delay Agreement was a rate limiter that caused the FDA to wait until November 30, 2011 to issue formal, written approval to Ranbaxy's ANDA.

372. The acts and practices of Pfizer alone, and of Pfizer and Ranbaxy working together, had the purpose and effect of restraining competition unreasonably and injuring competition by protecting Lipitor from generic competition for a substantial period of time until the present day, and for some time into the future. Defendants' actions allowed Pfizer to maintain a monopoly and exclude competition in the market for atorvastatin calcium, to the detriment of Giant Eagle.

373. As a direct and proximate result of some or all of the Defendants' overarching anticompetitive scheme, Ranbaxy or one or more other generic competitors would have begun selling AB-rated generic versions of Lipitor sooner than November 30, 2011, when Ranbaxy launched. As a direct and proximate result of the Defendants' overarching anticompetitive scheme, in whole or in part, Ranbaxy or one or more generic competitors would have launched generic Lipitor earlier than they did.

374. Ranbaxy and the other ANDA applicants seeking to market generic Lipitor had extensive experience in the pharmaceutical industry, including in obtaining approval for ANDAs, manufacturing commercial launch quantities adequate to meet market demand,

marketing generic pharmaceutical products, and paying and receiving consideration for selective waiver and/or relinquishment of 180-day first-to-file marketing exclusivities.

375. As a result of the delay in generic Lipitor competition brought about by Defendants' overarching anticompetitive scheme, in whole or in part, Giant Eagle paid more for atorvastatin calcium than it would have paid absent Defendants' illegal conduct.

376. Typically, generic versions of brand-name drugs are initially priced significantly below the corresponding branded drug to which they are AB-rated. As a result, upon generic entry, some or all of the direct purchases of branded drugs are rapidly substituted for generic versions of the drug. As more generic manufacturers enter the market, prices for generic versions of a drug predictably plunge even further because of competition among the generic manufacturers, and correspondingly, the brand name drug continues to lose even more market share to the generics.

377. This price competition enables all direct purchasers of the drugs to: (a) purchase generic versions of a drug at a substantially lower price and/or (b) purchase the brand name drug at a reduced price. Consequently, brand name drug manufacturers have a keen financial interest in delaying the onset of generic competition, and direct purchasers experience substantial overcharges from that delay.

378. If generic Lipitor competitors had not been unlawfully prevented from earlier entering the market and competing with the Defendants, and if Pfizer had not taken steps to suppress generic substitution after November 2011, Giant Eagle would have paid less for atorvastatin calcium by (a) substituting purchases of less-expensive AB-rated generic Lipitor for its purchases of more-expensive branded Lipitor, (b) paying less for its remaining branded

Lipitor purchases, and (c) purchasing generic Lipitor at lower generic prices than it actually paid, and/or (d) purchasing more generic Lipitor and less branded Lipitor.

379. Moreover, due to Defendants' conduct, other generic manufacturers were discouraged from and/or delayed in developing generic versions of Lipitor.

380. Thus, Defendants' unlawful conduct deprived Giant Eagle of the benefits of competition that the antitrust laws were designed to ensure.

X. ANTITRUST IMPACT

381. During the relevant period, Giant Eagle (or its assignor) purchased substantial amounts of Lipitor from Pfizer. After generic entry, Giant Eagle purchased substantial amounts of generic Lipitor from Ranbaxy. As a result of Defendants' illegal conduct, Giant Eagle was compelled to pay, and did pay, artificially inflated prices for its atorvastatin calcium requirements. Those prices were substantially greater than the prices Giant Eagle would have paid absent the illegal conduct alleged herein, because: (1) the price of brand-name Lipitor was artificially inflated by Defendants' illegal conduct and/or (2) Giant Eagle was deprived of the opportunity to purchase lower-priced generic versions of Lipitor sooner.

382. As a consequence, Giant Eagle has sustained substantial losses and damage to its business and property in the form of overcharges. The full amount and forms and components of such damages will be calculated after discovery and upon proof at trial.

XI. CLAIMS FOR RELIEF

CLAIM I: VIOLATION OF 15 U. S. C. § 2
(Monopolization and monopolistic scheme against Pfizer only)

383. Giant Eagle hereby incorporates each preceding and succeeding paragraph as though fully set forth herein.

384. This claim is asserted against the Pfizer Defendants only.

385. At all relevant times, Pfizer possessed substantial market power (i.e., monopoly power) in the relevant market. The Pfizer Defendants possessed the power to control prices in, prevent prices from falling in, and exclude competitors from the relevant market.

386. Through the overarching anticompetitive scheme, including the Delay Agreement, sham citizen petition, and other misconduct, as alleged herein, the Pfizer Defendants willfully maintained their monopoly power in the relevant market and slowed the loss of their monopoly power using restrictive or exclusionary conduct, rather than by means of greater business acumen, and injured Giant Eagle thereby.

387. It was Pfizer's conscious object to further its dominance in the relevant market by and through the overarching anticompetitive scheme.

388. As a direct and proximate result of the Pfizer Defendants' illegal and monopolistic conduct, as alleged herein, Giant Eagle suffered antitrust injury as alleged above.

CLAIM II: VIOLATION OF 15 U. S. C. § 1
(Agreement in restraint of trade against all Defendants)

389. Giant Eagle hereby incorporates each preceding and succeeding paragraph as though fully set forth herein.

390. This claim is asserted against all Defendants.

391. In 2008, Pfizer and Ranbaxy entered into the Delay Agreement, and Ranbaxy thereby joined the Pfizer's overarching anticompetitive scheme as a co-conspirator. The Delay Agreement is and was a contract, combination and/or conspiracy that substantially, unreasonably, and unduly restrained trade in the relevant market, the purpose and effect of which were to: (a) allocate all sales of atorvastatin calcium in the United States to the Pfizer Defendants until November 30, 2011; (b) prevent the sale of any generic version of atorvastatin calcium in

the United States until November 30, 2011; and (c) fix the price which Giant Eagle would pay for atorvastatin calcium.

392. Under the Delay Agreement, Pfizer paid Ranbaxy compensation, and in exchange Ranbaxy agreed to, and did, delay introduction of its generic Lipitor.

393. The Delay Agreement harmed Giant Eagle as set forth above.

394. The Delay Agreement covered a sufficiently substantial percentage of the relevant market to harm competition.

395. Pfizer and Ranbaxy are each *per se* liable for the creation, maintenance, and enforcement of the Delay Agreement.

396. Alternatively, Pfizer and Ranbaxy are liable for the Delay Agreement under a "quick look" and/or rule of reason standard.

397. There is and was no legitimate, nonpretextual, procompetitive business justification for the Delay Agreement, nor for the compensation to Ranbaxy under the Delay Agreement, that outweighs its harmful effect. Even if there were some conceivable such justification, the Delay Agreement is and was broader than necessary to achieve such a purpose.

398. As a direct and proximate result of Pfizer's and Ranbaxy's anticompetitive conduct, as alleged herein, Giant Eagle suffered antitrust injury as alleged above.

CLAIM III: VIOLATION OF 15 U. S. C. § 2
(Conspiracy to monopolize against all Defendants)

399. Giant Eagle hereby incorporates each preceding and succeeding paragraph as though fully set forth herein.

400. This claim is asserted against all Defendants.

401. Through the overarching anticompetitive scheme, including the Delay Agreement, sham citizen petition, and other misconduct as alleged herein, Pfizer and Ranbaxy conspired to maintain and enhance Pfizer's monopoly power in the relevant market.

402. Pfizer and Ranbaxy knowingly and intentionally conspired to maintain and enhance Pfizer's monopoly power in the relevant market.

403. Pfizer and Ranbaxy specifically intended that the overarching anticompetitive scheme would maintain Pfizer's monopoly power in the relevant market, and injured Giant Eagle thereby.

404. Pfizer and Ranbaxy each committed at least one overt act in furtherance of the conspiracy.

405. As a direct and proximate result of Pfizer's and Ranbaxy's illegal and monopolistic conduct, Giant Eagle suffered antitrust injury as alleged above.

CLAIM IV: VIOLATION OF 15 U. S. C. § 2
(Attempted monopolization against Pfizer only)

406. Giant Eagle hereby incorporates each preceding and succeeding paragraph as though fully set forth herein.

407. This claim is asserted against the Pfizer Defendants only.

408. The Pfizer Defendants, through their overarching anticompetitive scheme, including the Delay Agreement, sham citizen petition, and other misconduct as alleged herein, specifically intended to maintain monopoly power in the relevant market and to slow down the loss of that monopoly power. It was Pfizer's conscious objective to control prices and/or to exclude competition in the relevant market.

409. The natural and probable consequence of the Pfizer Defendants' overarching anticompetitive scheme, which was intended by, and plainly foreseeable to, Pfizer, was to control prices and exclude competition in the relevant market, to the extent it did not succeed.

410. There was a substantial and real chance, a reasonable likelihood, and/or a dangerous probability that Pfizer would succeed in and achieve its goal of maintaining monopoly power in the relevant market.

411. As a direct and proximate result of the Pfizer Defendants' illegal and monopolistic conduct, Giant Eagle suffered antitrust injury as alleged above.

CLAIM V: VIOLATION OF THE OHIO VALENTINE ACT
(Agreement in restraint of trade against all Defendants)

412. Giant Eagle hereby incorporates each preceding and succeeding paragraph as though fully set forth herein.

413. Defendants conspired to restrict, forestall and prevent competition for sales of atorvastatin calcium in violation of Ohio's Valentine Act, Ohio Rev. Code §§1331.01, 1331.04, 1331.06 and 1331.08. This claim is asserted against all Defendants.

414. In 2008, the Pfizer and Ranbaxy entered into the Delay Agreement, and Ranbaxy thereby joined Pfizer's overarching anticompetitive scheme as a co-conspirator. The Delay Agreement is and was a contract, combination and/or conspiracy that substantially, unreasonably, and unduly restrained trade in the relevant market, the purpose and effect of which were to: (a) allocate all sales of atorvastatin calcium in the United States to the Pfizer Defendants until November 30, 2011; (b) prevent the sale of any generic version of atorvastatin calcium in the United States until November 30, 2011; and (c) fix the price which Giant Eagle would pay for atorvastatin calcium.

415. Under the Delay Agreement, Pfizer paid Ranbaxy compensation, and in exchange Ranbaxy agreed to, and did, delay introduction of its generic Lipitor.

416. The Delay Agreement harmed Giant Eagle as set forth above.

417. The Delay Agreement covered a sufficiently substantial percentage of the relevant market to harm competition.

418. Pfizer and Ranbaxy are each *per se* liable for the creation, maintenance, and enforcement of the Delay Agreement.

419. Alternatively, Pfizer and Ranbaxy are liable for the Delay Agreement under a "quick look" and/or rule of reason standard.

420. There is and was no legitimate, nonpretextual, procompetitive business justification for the Delay Agreement, nor for the compensation to Ranbaxy under the Delay Agreement, that outweighs its harmful effect. Even if there were some conceivable such justification, the Delay Agreement is and was broader than necessary to achieve such a purpose.

421. As a direct and proximate result of Pfizer's and Ranbaxy's anticompetitive conduct, Giant Eagle suffered antitrust injury as alleged above.

XII. DEMAND FOR JUDGMENT

WHEREFORE, Giant Eagle prays for judgment against Defendants and for the following relief:

A. A judgment for three times its actual damages, as determined by a jury at trial;

B. Permanent injunctive relief enjoining Defendants from continuing their unlawful conduct and requiring them to take affirmative steps to dissipate the effects of their prior conduct;

C. The costs of this suit, including reasonable attorneys' fees as provided by law; and

D. Such other and further relief as the Court deems just and appropriate.

XIII. JURY DEMAND

Giant Eagle demands a trial by jury of all issues so triable.

Dated: March 1, 2013

Respectfully submitted,

/s/ Moira Cain-Mannix

Bernard D. Marcus
PA ID No. 01293
Moira Cain-Mannix
PA ID No. 81131

MARCUS & SHAPIRA LLP
One Oxford Centre, 35th Floor
301 Grant Street
Pittsburgh, Pennsylvania 15219
Telephone: (412) 471-3490
Facsimile: (412) 391-8758

Counsel for Plaintiff, Giant Eagle, Inc.